JULY

THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION

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THE JOURNAL
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Original Communications

AMALGAM FILLINGS, WITH SOME SUGGESTIONS AS TO THEIR PRACTICAL USE*

By ROBERT E. BLACKWELL, D.D.S., M.S., F.A.C.D., Chicago, Ill.

IN April, 1928, under a cooperative agreement between the National Bureau of Standards and the American Dental Association, work was begun on a study of dental amalgams. Previous to this time, some valuable studies had been made by Wilmer Souder and others of the Bureau, which were reported in the *Dental Cosmos* for 1920. The reports of the more recent studies undertaken in the Bureau have been made from time to time in THE JOURNAL.

The findings of Dr. Souder and his associates are important and, in some respects, mean so much to the profession that a brief review of some of them at this time would appear to be worth while.

The studies undertaken in the Bureau have included (1) the composition of dental amalgam alloys, (2) dimensional changes with temperature, (3) dimensional changes with time, (4) flow under compression, (5) crushing strength, (6) tensile strength, (7) a comparison of mechanical and hand-mixed amalgamation and (8) the effect of packing pressures on setting changes.

These studies have made it possible, and shown it to be desirable, for the American Dental Association, in cooperation with the Bureau, to make tentative specifications for dental amalgam alloys. These specifications, as made and published in THE JOURNAL January, 1930, were based on Federal specification No. 356, which had been used for some time in connection with the purchase of alloys for the U. S. Government.

Among the detailed requirements of the tentative specifications as they now stand are the following:

1. Chemical composition. This shall be within the following limits: silver, from 65 to 70 per cent; tin, from 26 to 29 per

*Read before the Section on Operative Dentistry at the Midwinter Clinic of the Chicago Dental Society, Feb. 4, 1931.

cent; copper, from 3 to 6 per cent; zinc, from 0 to 2 per cent.

2. Ultimate compressive strength. The average ultimate compressive strength values of three or more tests shall not fall below 2,500 kg. per square centimeter, or approximately 35,500 pounds per square inch. (This reduced to an area of one-eighth inch would amount to about 555 pounds.)

3. Flow. Specimens subjected to constant pressure shall not show more than 5 per cent flow (shortening in length) in twenty-four hours.

4. Setting changes. Twenty-four hours after amalgamation, the length shall have increased between 1 and 10 microns per centimeter. If, at any time during the test, a contraction of more than 4 microns per centimeter is detected, the alloy shall be rejected.

The first alloys examined were tested to determine whether or not they complied with Federal specification No. 356. Twenty-six alloys were studied and a report made at the Minneapolis meeting in August, 1928.[1] Out of the number which were examined, only six met the requirements and were judged satisfactory. Fifteen of the alloys examined were shown to comply with the specification for chemical composition; that is, nine out of fifteen alloys which met the requirements for composition were not up to standard in regard to dimensional change, or in percentage of flow or in some other item or items in the specification. Six of these nine were unsatisfactory in regard to setting changes and two were unsatisfactory in respect to flow.

These studies clearly showed that proper proportions of the chemical constituents of an alloy are not all that is required in "balancing" the alloy. Treatment in fusing, or cutting or annealing may so affect the physical qualities of two or more alloys made by the same formula that the finished articles will vary quite markedly in regard to dimensional change. In regard to this, Taylor[2] says, " . . . while no alloy failing to meet the composition standards may be expected to have satisfactory physical properties, the fact that an alloy may be acceptable as far as composition is concerned is no guarantee that its physical properties will also be acceptable. Physical tests indicate that only 40 per cent of the alloys meeting the chemical requirements were up to standard physically."

This report substantially supports the earlier conclusions of the late G. V. Black that while the silver-tin content should lie within quite definite limits, no fixed formula for a "balanced" alloy could be relied on unless the materials entering into the alloy were chemically pure and the methods of fusing and subsequent treatment were alike in every particular.

In 1929, further studies were made of forty-four marketed alloys. Of these, eighteen were found to be satisfactory. In connection with this report, Taylor says that several of the manufacturers have improved their products since the 1928 survey of amalgam alloy was published. In some cases, the improvement has not been enough to cause the materials to come up to specifications, but it shows a willingness on the part of the manufacturers to produce alloys of good quality.

The effect of these studies on the quality of work that is being done and will be done in the future cannot be

1. Taylor, N. O.: Survey of Amalgam Alloys: Report to Research Commission of American Dental Association, J.A.D.A., 16:590 (April) 1929.

2. Footnote, 1, p. 594.

estimated. Three years ago, comparatively few men were in a position to know what alloys were fit for use. The average dentist no doubt bought the alloy that his favorite dealer recommended. It might have been one of the six good ones, but just as likely it was not. During the last year, as a result of the continued work of our research associates in the Bureau, we have a larger number to select from. Anyone may turn to Table 1, pages 115 and 116, THE JOURNAL, January, 1930, and, in a few minutes, determine whether the alloy he has been using is a good one, and if it is not, he may select one that should give satisfactory results if properly handled.

Too much emphasis cannot be placed on the importance of this recent work, which has been so well done and which, coming from the Bureau of Standards, can be accepted without question as of the highest order and authenticity. Such a report on marketed materials could be made only by government investigators without arousing a great deal of indignation and charges of prejudice. No individual could afford to do this work. Dr. Black was urged to make a report on marketed alloys years ago, but wisely refused to mention the names of manufacturers.

Some altogether worthless alloys have been put on the market by manufacturers who have not had the instruments in their laboratories for making proper physical tests. Dentists have bought them and, in some cases, have paid high prices for these materials, believing them to be good, and then have been disappointed with the quality of the fillings. One alloy whose manufacturers claimed a 5 per cent gold content and for which an accordingly high price was paid (I believe it was $6 an ounce) was found to contain no gold, but was so high in tin content that it was unsatisfactory.

As a result of these reports, manufacturers are being forced to market only high-grade alloys, and I believe it safe to predict that all inferior materials will soon be brought up to standard or withdrawn from the market.

The use of poor materials is one reason that amalgam has fallen into disfavor with many dentists and with the public. But the data are now in our hands, and there is no longer any excuse for using inferior alloys.

The next point of importance which has been brought to our attention by these studies is the effect of manipulation on setting changes. Taylor[3] has reported on the effect of mechanical mixing and has shown that, in general, mechanical amalgamation gives an amalgam which shrinks more on setting than if the same material were hand mixed. This is of great importance from the standpoint of permanent adaptation, for, of course, an ideal amalgam should never shrink. It appears from Dr. Taylor's report that it is possible to employ mechanical amalgamation in the handling of certain alloys without bringing about excessive shrinkage, but the indiscriminate use of this method is inadvisable.

As the greater length of time consumed in hand mixing is such a small factor, not more than two minutes, it would seem that we are not justified in taking the risk of greater shrinkage caused by mechanical amalgamation. Furthermore, as such mechanical devices add their bit to the expense of operating and tend to clutter up an office more or less, it is my opinion that we may well dispense with them.

3. Taylor, N. C.: Mechanical Amalgamation: Progress Report of Research on Dental Materials, J.A.D.A., 16:583 (April) 1929.

In regard to hand mixing, time and grinding pressures are factors of importance. Souder[4] showed, in 1920, that prolonged mulling of the mass in the hand caused shrinkage.

Presumably as a result of his work, amalgams are mixed lightly in a mortar for one minute and mulled in the hand for two minutes, in the Bureau laboratories, unless more definite directions are given by the manufacturer. Heavy grinding pressure should be avoided as this may produce shrinkage. The mixing in the mortar should be done lightly to avoid heating the material, and plasticity should be secured by mulling in the hand.

In regard to the technic of filling the cavity, that is, packing pressure and adaptation, we are left somewhat in the dark as to the best procedure. Taylor has shown that light packing pressures cause slight expansion on setting and that heavy pressures may cause shrinkage. The accompanying table[5] illustrates this point.

EFFECT OF PACKING PRESSURES ON SHRINKAGE AND EXPANSION

How Packed	Change in in 24 hours (Microns per Centimeter)
Vibrated; no instrument used	+18.0
Vibrated; instrument used to condense lightly	+13.0
Pressure on plugger, 1.5 mm. in diameter	
2 pounds	+5.5
4 pounds	+2.0
6 pounds	+0.5
8 pounds	+0.0
Malleted to place	—1.0

4. Souder, W. H.: Technologic Paper No. 157.

5. Taylor, N. O.: Amalgam Technic: Dependable and Dangerous Practices, J.A.D.A., 17:1887 (Oct.) 1930.

In the mind of the practical operative dentist, the question immediately arising is how to obtain the best adaptation to cavity walls and not cause shrinkage which would nullify the effect of mechanical adaptation. According to this table, one may use 8 pounds packing pressure without doing harm. Many of us are in the habit of using more than this even though the opinion has been expressed that from 3 to 4 pounds represents the average pressure obtainable on small pluggers. I have made many tests of my finger power with the Black manudynamometer and have recorded 20 pounds pressure with the pen grasp. Fifteen pounds pressure was easily produced again and again, which indicated that this pressure could be continued throughout the packing of a fairly large cavity. For the final pressure with large pluggers over the entire mass, it has been customary to employ both hands. In this case, the amount of pressure may be 20 pounds or more. Does this pressure cause shrinkage? According to the table just quoted, it could be expected to. But Taylor goes on to say that in the few cases in which the manufacturers are aware of this effect (of packing pressures), their products have been so balanced and annealed as to show expansion under the pressures commonly obtained in the mouth. Manufacturers should give very definite directions in regard to methods of mixing and packing, and we dentists should insist on being furnished with this information and then make practical use of it.

There is some evidence that the reports from the Bureau are forcing manufacturers to give directions for handling their materials, but there is still room for a great deal of improvement. I recently studied the directions found in the packages of five different high-grade alloys,

all of which are extensively used. Not one gave directions that would satisfactorily cover all of the following: (1) the proportion of alloy and mercury; (2) the best method of mixing the amalgam; (3) the manner of packing with respect to pressure.

The profession is in urgent need of information on adaptation of amalgam, and it is rather surprising, in view of the excellent work that has been done on amalgam in the Bureau, that more attention has not been given to this subject. A search of their reports reveals little of value along this line.

The nearest approach to definite directions for packing the cavity that I have found in articles published by the Bureau are in the October number of THE JOURNAL.[5] Here Taylor says:

> The first portions of alloy placed in a cavity should be soft and plastic. Condensation should take place first at the edges, followed by wedging pressure, exerted in the central portions of the cavity. Alloy should be added in small pieces with some of the mercury expressed, but care should be taken to leave the surface of the filling wet with mercury at all times during the packing operations.

The emphasis here appears to be on condensation rather than adaptation. For one who is familiar with working amalgam, it is difficult to understand how condensation at the edges with small instruments (definite sizes are not given), followed by wedging pressure in the center of the mass, will produce and maintain adaptation to cavity walls when the mass is in so plastic a condition as it must be in when wet with mercury.

During his lifetime, there probably was no one who had a more thorough knowledge of amalgam than the late G. V. Black. He was familiar with the requirements for composition, fusing, and annealing; and to his laboratory studies was added many years of practical experience. Dr. Black was convinced that the mass should be grasped as a whole, as nearly as possible, and compressed into the cavity. He says, "The whole principle of making perfect work is contained in the one word, compression."[6]

The technic of packing the cavity as described by Dr. Black is briefly as follows:

> Pluggers and packing instruments should be as large as can well be used in the cavity, so as to grasp and compress the amalgam as a whole as nearly as possible in the first instance, and then used with all the power of the hand, and with just a slight shaking motion, which tends to settle the amalgam into any irregularities or angles of the cavity. The rope of amalgam should be broken into bits the size desired before beginning so that they can be handled quickly with the pliers and placed in position. Then each piece is caught by the broad plugger point and immediately condensed. A small point should always be in readiness on the tray for special condensation about the walls and into the angles or any points that the large instruments may not reach well. This should be done on the same principle as the stepping of the instrument in packing gold to secure perfect adaptation to walls and margins. That is, the stepping should be *from the mass of the filling already condensed towards the walls, finishing at the walls and wedging the last between the walls and the portion already packed.*
>
> In using small instruments about the walls and angles, the force should be sufficient to pack the amalgam, but not so great as to force the instrument into the already packed material and cause it to move. It requires some care to learn this and to use force judiciously. In the main work with the large instrument, the whole power of the hand will not be too much, but with small points it is easy to force the previously packed material out of position.
>
> The packing should be continued carefully and rapidly, and the filling should be built up evenly, until the cavity is full and more than full. The amalgam should be heaped up

6. Black, G. V.: Operative Dentistry, Vol. 2, Chicago: Medico Dental Publishing Co., 1908.

over the margin. Then, with the largest instrument possible, all the pressure the hand can give should be applied, shaking very slightly, compressing the mass as a whole as much as possible.[7]

This is a very different technic from the one suggested by Dr. Taylor. Dr. Black's word virtually has been considered law in regard to amalgam. His technic has been employed by thousands of dentists all over the world for many years and it has given excellent results. For these reasons, if our research associates in the Bureau believe that radical changes in technic are necessary in order to get the best results with the material, a detailed technic based on sound evidence that better adaptation can be obtained with its use should be offered the profession. This point is much too important not to be dealt with fully by those who are best equipped to make such a study.

A FEW REASONS THAT AMALGAM FILLINGS FAIL

It has been generally recognized and remarked on by dentists everywhere that amalgam is a much abused material. Also, it frequently has been said that amalgam, in spite of its abuse, possibly saves more teeth than all other filling materials combined. As to the former statement, there is much evidence in support of it; in regard to the latter, some question arises as to the meaning of saving teeth. Certainly, amalgam has abundantly demonstrated its ability to save carious teeth; that is, a well manipulated amalgam can be expected to seal a cavity and prevent further decay on the surface or surfaces involved. The abuse which has been mentioned frequently applies more to the teeth and to the patient than to the amalgam. The amalgam may do its part well enough and stop the ravages of caries, but the dentist's failures in cavity preparation, in contour and contact, in trimming and polishing, may contribute to the eventual loss of the teeth.

Amalgam may well be considered one of the permanent filling materials. Dr. Black rated it as a close second to gold foil in this respect. Certainly, we owe our patients as much consideration in preparing cavities for amalgam as for gold foil or gold inlays. There usually is no more excuse for the recurrence of decay at the gingival margins or the buccogingival or linguogingival angles of proximal amalgam fillings than for the recurrence of decay around fillings of other materials, provided the cavities are properly extended for prevention. Well-placed fillings in cavities which have been properly outlined for extension for prevention will give good results whether the material is amalgam or gold.

Another serious fault in the preparation of cavities for amalgam fillings is a failure to make adequate resistance form. Many dentists depend on undercuts for retention and stability. Proximal cavities are frequently prepared without an occlusal step of sufficient size to give this desired stability. The undercuts in the buccal and lingual walls retain the filling; but, under heavy stress of mastication, the filling may move because it is not well supported. This invites the recurrence of decay and not infrequently results in exposure of the pulp by caries as the progress of decay is usually very rapid under a leaking filling.

The practice of utilizing natural undercuts for the retention of amalgam has frequently resulted in frail, unsupported enamel walls not being cut down but allowed to stand until broken down by the stress of mastication at some subse-

7. Footnote 6, p. 323.

quent time. Amalgam, because of the very nature of this plastic material, is capable of being adapted to frail walls without shattering them, but good judgment and good operating require that such frail walls be cut down and that the filling be built over the part removed in such a way as to take the stress of mastication.

Probably the most severe and frequent criticism to which amalgam as a filling material has been subjected is due to the presence of carious material found under the fillings. The complaint is commonly heard that there always is decay under amalgam. To those who have done careful operative work and watched the results critically year after year, it is perfectly evident that such criticism should be directed at the thousands of dentists who use and misuse amalgam and not at the material itself. Some dentists apparently are careless in regard to the removal of carious dentin; a few are ignorant in respect to the requirements for its removal, and some lack courage. Perhaps all of these faults are to be found in the make-up of the dentists who use amalgam extensively. Whatever the excuse may be for failure to remove carious material, the blame cannot rightfully be laid at the door of amalgam.

Carelessness or inattention to the preparation of the enamel walls is another contributing factor in amalgam failure. Enamel should rest on sound dentin. The enamel walls and margins should be planed just as carefully as though the preparation were for a gold inlay. A sharp bevel of the occlusal margin should be avoided as amalgam is lacking in edge strength, but the gingival margin may and should be planed smooth and beveled with the appropriate gingival margin trimmers.

Failure to freshen cavity walls before filling the cavity and to exclude moisture from the cavity during the period of packing are probably responsible for many failures with amalgam. Black[8] has clearly shown that saliva drying in a cavity would leave a residue of soluble salts on the walls of the cavity which would be dissolved by the fluids of the mouth after the filling was placed. Such a film of soluble material, invisible to the unaided eye, would cause a leak at the margins which would admit moisture to the cavity. If moisture only were admitted to the cavity, perhaps nothing more serious than a discoloration of the tooth due to the presence of hydrogen sulphid would occur; but if acid-forming bacteria could colonize at such a leaking margin, the result would be a failure. The leaking margin would soon be carious.

No amount of packing pressure on amalgam can possibly drive all the moisture from a cavity. Moisture should be absorbed and the last traces of it removed by planing with instruments just before placing the filling.

During the packing of the cavity, moisture interferes with the cohesion of the mass. Taylor[9] reports that the contact of saliva with amalgam during the filling operation decreases the crushing strength about 20 per cent and causes brittleness.

Few proximal amalgam fillings in any of the back teeth can be entirely successful unless provision has been made for restoring full contour and for making a proximal contact. Many operators who use good alloys and have a reliable technic for mixing and packing fail miserably in their efforts or lack of efforts in producing contour and contact. The employment of any filling should be a prophylactic meas-

8. Footnote 6, p. 324.
9. Footnote 5, p. 1888.

ure in the prevention of further decay and the protection of the supporting structures from injury. A filling should restore the tooth to its full use in mastication and maintain that condition. When proximal contour and contact are not restored, food is almost certain to pack into the interproximal space, and the accumulated débris will cause a recurrence of decay at the gingival margin or serious injury to the peridental membrane. In such cases, the secondary injury is more serious than the original decay. These facts are quite generally known by the profession, but the apparent disregard of the serious consequences of weak or flat contacts makes it necessary to call attention again and again to these prevailing conditions. Where there is good occlusion, separation may be gained by packing the cavity with baseplate guttapercha and allowing the patient to masticate on this for a few days. But the Perry separator offers a quick and certain way of obtaining adequate separation for our needs. To one who has familiarized himself with the use of this instrument, it is indispensable.

Many operators do not trim or polish amalgam fillings. An excess of material at the gingival margin, filling or nearly filling the interproximal space, is not an occasional find, but, rather, a common occurrence. A few years ago, Arthur D. Smith gave a clinic on gold inlays before this society in which he exhibited a large number of extracted teeth containing proximal amalgam fillings. Our attention was called to the fact that while the occlusal surfaces of these fillings were polished, the proximal surfaces were sadly in need of trimming. The cause of failure and loss of the tooth was plainly seen on each specimen shown. This exhibit was used as an argument against the use of amalgam and for the use of gold inlays. In my opinion, it was a serious indictment of the dentists who filled the cavities, but not of amalgam as a filling material.

There has been an attempt on the part of some observers to blame such conditions of excess material on the flow of the amalgam. It is said that the continual pressure of mastication on the occlusal marginal ridge results in the movement of the proximal portion of the filling into the interproximal space. Perhaps where low-grade alloys have been used, this might be true. But wherever high-grade material has been used, I have never observed any change in the filling that could be attributed to the flow of the amalgam.

It is a matter of common observation that comparatively few operators polish amalgam fillings. It is also observed that most operators give considerable attention to polishing gold inlays and gold foil fillings. The reason for this difference in treatment of fillings that are considered permanent restorations is a little hard to understand. Presumably, many men consider the polishing operation an unnecessary effort and believe that the fee for the filling does not justify the expenditure of time for polishing. In a relatively few cases, failure to polish may be due to the difficulties involved in arranging a second appointment. Usually, when a job is incomplete, if reasons are asked for, excuses are offered. This is undoubtedly true in the matter of unpolished amalgam fillings.

An unpolished amalgam filling certainly is not so good a filling as one that is well polished. Adaptation of amalgam at the margins can be improved in much the same way that marginal adaptation of gold inlays can be improved, although amalgam and cast gold are not identical in physical qualities. Burnishing toward the margins before the amalgam has set is not beneficial and may result in reducing the edge strength by carrying an excess of mercury toward the margins and

weakening the filling where the greatest strength is desired.

Amalgam fillings that are thoroughly polished do not discolor, that is, turn black or brown, so readily as unpolished fillings. The fillings may not retain their luster, but will turn a dull gray, which in the back teeth is not particularly objectionable.

INDICATIONS FOR AMALGAM; OCCLUSAL AND GINGIVAL THIRD CAVITIES

Probably, many of us here prefer to use gold for the usual filling restorations in the back teeth. But in occlusal cavities, amalgam has proved to be of such great value, running a close second to gold foil in serviceability, that it should be used extensively.

According to the usual order of occurrence, pit and fissure cavities are the first to appear. The permanent teeth erupting at a time when susceptibility to caries is pronounced may need filling almost immediately. Some time later, possibly from three to five years, proximal cavities are very likely to be found. In the mouths of persons very susceptible to caries, this is to be expected. In the first permanent molars, for example, occlusal decay may be discovered at between 8 and 10 years of age, while proximal cavities in the same teeth may not appear until at 14 or 15 years of age. It is desirable to so plan the operations, especially for people of limited means, that the patient will eventually have the best restorations that it is possible for us to make at a cost which is not excessive. It will often happen that the occlusal filling which is placed at 8 or 10 years of age will have to be removed before the patient is 20, in order to gain stability and retention for a proximoclusal filling. In case the occlusal cavity originally was filled with gold and the proximoclusal restoration also is to be of gold, the cost to the patient will be considerably greater than is necessary. A good amalgam filling placed in the occlusal cavity could be expected to last indefinitely, certainly until its removal was made necessary by the discovery of proximal decay in the same tooth, and the cost of the amalgam may be one-half or one-third that of the gold filling or gold inlay.

Gingival third cavities, although usually appearing some years later than occlusal cavities, again offer a splendid opportunity for the use of amalgam. These cavities should be as shallow as we can make them, particularly for young people, and for that reason should be undercut for retention. Gold inlays in these positions require deeper cutting for retention and for this reason are less desirable. The appearance of several gingival third cavities simultaneously is a sure sign of severe susceptibility to caries and calls for the maximum of extension for prevention and great care and fine work in order that caries may be kept under control. Gold foil probably makes the most serviceable filling, but it cannot be used so extensively as we might wish on account of the difficulties of access in the back teeth. Again, its cost often is prohibitive. But amalgam fillings, well placed and well polished, can be depended on to give splendid service.

PROXIMAL CAVITIES IN BICUSPIDS AND MOLARS

It has been mentioned in a previous paragraph that proximal contour and contact are paramount considerations in making proximal restorations. The gold inlay lends itself so nicely to the perfection of these conditions that many operators have come to use cast gold to the exclusion of every other means of restoration. In my opinion, the extensive use of

gold inlays in proximoclusal cavities is not to be deplored, but their exclusive use is a great mistake.

From the economic point of view alone, it is a mistake not to use amalgam. A proximal amalgam filling which is made with the rubber dam in position and with separation and with a good matrix, well secured, can be inserted for not more than half the cost to the patient of the equivalent gold inlay, because the time required for the amalgam filling is so much less than the total chair and laboratory time required for the gold inlay. In making this statement, I am not regarding amalgam as a cheap material but rather as a high-grade material meriting careful work and a good fee. For patients of limited means, amalgam is not used for filling proximal cavities so much as it should be.

On the other hand, in attempting to keep the cost of service within reach of our less prosperous patients, it occasionally happens that we would spend so much time in overcoming the difficulties met with in operating, such as the adapting of the rubber dam, matrix and separator, that it would be better to make gold inlays, possibly for the same price as the amalgam fillings, than to consume so much time and energy in placing the amalgam.

USE OF AMALGAM IN THE DECIDUOUS TEETH

Amalgam is probably the best material at our command for filling the deciduous molars. For occlusal cavities, it makes a well-nigh perfect filling. In proximal cavities, much more difficulty, naturally, is encountered; nevertheless, it should be used extensively instead of cements, because of its ability to maintain proximal contact. This subject more properly belongs in a paper on the care of children's teeth.

In closing, I should like to refer again to G. V. Black, who, in his final word on amalgam in his "Operative Dentistry," says:

> **The idea that amalgam is a cheap filling, to be done quickly "in any old way" should be discarded forever. The dentist should have the same pay for time in making amalgam fillings as in making gold fillings, and take the time to do it well. If he does his duty as well, the service to his patient in proportion to the time employed will be just as valuable.**
>
> **Fillings properly made of the modern amalgams are a close second to gold (foil) in their durability and in their protection against the recurrence of caries. The difference is that the amalgam filling is the more difficult to make perfectly. It is more difficult to learn the manipulation, and, even then, it is more difficult to secure perfect results regularly, filling after filling. . . .**[9]

9. Footnote 6, p. 329.

NEW LIGHT ON THE CONTROL OF DENTAL CARIES AND THE DEGENERATIVE DISEASES*

By WESTON A. PRICE, D.D.S., M.S., F.A.C.D., Cleveland, Ohio

IN man's universal struggle for life, the emphasis has been rapidly passing from the rôle of the invading organism to the factors which determine the capacity of the host to maintain defense against the invader. With this is coming a new view of the fundamental contributing factors to the degenerative processes, including dental caries, which, while usually associated with evidence of bactrial invasion, are coming rapidly to be viewed as being, in large part, nutritional in origin. The emphasis of various workers in different fields has been placed on different ones of the few already identified vitamins. I have interpreted the available data as indicating the presence of a group of activators not limited to the known vitamins. For the past eight years, I have been presenting research reports indicating consistent and regular progress in the control of dental caries in which various of the contributing factors have been discussed in connection with mineral utilization and defense against disease.[1]

These studies have provided a large mass of data indicating that dental caries is primarily a nutritional disturbance, though influenced by local environmental conditions, and that it is directly related in its etiology to some other degenerative processes. The materials that I have briefly utilized for treatment and recom-

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1. Price, W. A.: Fundamentals Suggested by Recent Researches for Diagnosis, Prognosis and Treatment of Dental Focal Infections, J. A. D. A., 12:1468 (June) 1925; Newer Knowledge of Calcium Metabolism in Health and Disease, with Special Consideration of Calcification and Decalcification Processes, Including Focal Infection Phenomena, ibid., 13:1765 (Dec.) 1926; Relation of Light to Life and Health, Indus. & Eng. Chem., July, 1926; Calcium Metabolism Studies, A. Raising of Serum Calcium by Topical Applications of Raw and Activated Cod Liver Oil, B. Disturbances Associated with Active Dental Caries of Childhood and Pregnancy; Am. J. Dis. Child., 33:78 (Jan.) 1927; Calcium, Its Activation, Utilization and Metabolism; J. A. D. A., 15:729 (April) 1928. Some New Fundamentals for the Prevention of Dental Disease, with Special Consideration of Calcification and Decalcification Processes, Dent. Cosmos, 71:145 (Feb.) 1929. Calcium Metabolism Studies on Nature and Rôle of Activators; Researches on Fundamentals for Prevention of Dental Disease; J. A. D. A. 16:265 (Feb.) 1929; New Fundamentals for Treatment and Prevention of Dental Disease Based on Calcium Utilization and Disturbance, with Special Consideration of Factors Determining When Infected Tooth Becomes Liability, ibid., 16:456 (March) 1929; Calcium and Phosphorus Utilization in Health and Disease. I. Rôle of the Activators for Calcium and Phosphorus Metabolism. II. Nature and Source of Calcium and Phosphorus Activators. III. Seasonal Variations in Butter-Fat Vitamins and Relation to Seasonal Morbidity, Cert. Milk, October, November, December, 1929; Dominion Dent. J., 41:315 (Oct.), 351 (Nov.) 1929; Why the Annual Pilgrimages to the Hospitals? Your Health, March, 1930.

mended have included cod-liver oil, raw and activated, and activator concentrates from dairy products, together with adaptations of the diet.

The studies that I shall report in this paper include a review of the clinical results obtained through the administration of special activators taken in capsule form by an experimental group of more than 200 individuals, with a comparison with other individuals living under the same general conditions and in the same age periods who did not receive special vitamins. I have divided these individuals according to age into ten-year period groups for six decades. While it is clearly very difficult to control conditions in groups that are not under hospitalization, the differences in the groups are so great as to absorb all possible errors and leave a wide margin. The data are shown in graphic form in Figure 1, in which the dotted line shows by the elevation of the columns the number of cavities per person in the group not receiving the vitamin capsules, and the solid line shows the number of cavities per person in the individuals receiving capsules. In the entire group of all ages for the period covered, which was, in most cases, from September and October, 1929, to May and June, 1930, there were ten times as many cavities in the individuals not receiving capsules as in those receiving them, and, for the individual groups, the figures were as follows: for those from 1 to 10 years of age, inclusive, the ratio was 0:3; from 11 to 20 years of age, 1:40; in other words, in twenty individuals who received the capsules, there were only three cavities that developed in that period, whereas, in twenty similar individuals not receiving the capsules, there were disclosed 141 cavities. In the age groups from 21 to 30, the ratio was 1:6; for those from 31 to 40, 1:2.4; from 41 to 50, 0:1.0; from 51 to 60, 0:2.0. It is of interest to note that, with the continued research on and testing of activators, the percentage of ratios has progressively increased. For example, in two groups that were carried through the year 1928-1929, one with

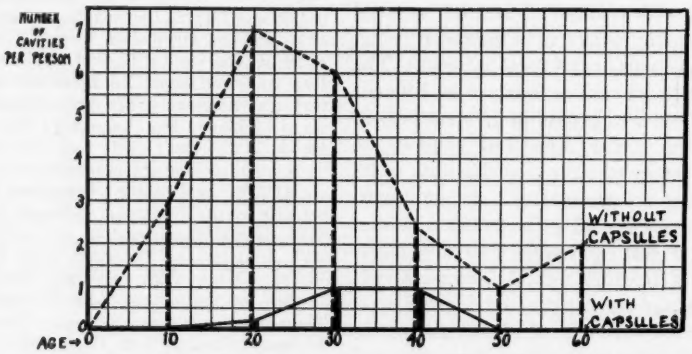

Fig. 1.—Effect of vitamin capsules on dental caries by ten-year age groups. Progress in the control of dental caries is shown by the difference in the number of cavities which have developed per individual in the group receiving special activators as compared with those in the group not receiving them. For all ages, this difference is 1 to 10 and the "teen" ages, 1 to 40. Broken line, capsules; solid line, no capsules.

treatment and the other without, the former had 4.85 cavities per person and, in 1929, 4.73 per person. This group contained forty-nine patients. In another group which was selected from the available cases and consisted of those who had an exceedingly large number of cavities, namely, an average of 8.02 cavities per person for 1928 when not receiving treatment, the cavities dropped to 1.40 per individual when the patients received special activators in capsule form. In a previous report,[1] I presented data on fifteen individuals between the ages of 12 and 18, inclusive, in which nine individuals not receiving treatment had 5.50 cavities per person and those individuals receiving a group of pregnancy cases, one patient without treatment had nine cavities, and one with treatment, one cavity.

While the improvement in dental conditions has been marked, it is of great importance that the practically universal report of all the patients under special treatment has been improvement in physical well-being, reduction of colds and increased energy capacity. A large number of clinical conditions which were considered as diseases greatly improved or disappeared, which suggested that, in part, and probably in large part, they also were symptoms of nutritional imbalances. The association of these various phenomena has strongly indicated that we are dealing

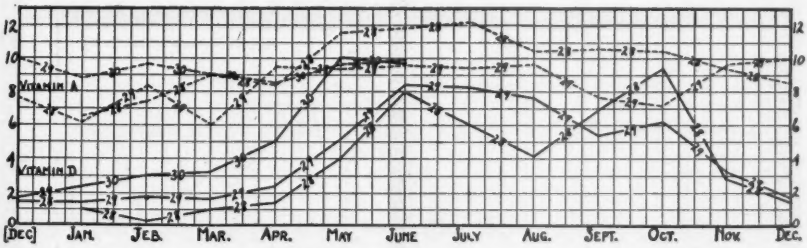

Fig. 2.—Comparison of vitamins A and D in butter by months for three years (1928, 1929, 1930). The levels of vitamins A and D as determined by chemical tests of the samples for the same five-state area for two whole years and six months of this year show, in general, the same rise in the early summer and autumn, with a depression in midsummer and a severe depression for winter and spring.

treatment did not have a single cavity. In the group attending boarding schools, four without treatment had an average of 8.50 cavities per person, and those receiving treatment, but living under the same conditions, had no cavities in four individuals. In one case, there were two brothers, the one taking capsules and the other not taking them. The one taking the capsules had no cavities and the one not taking them had five. These individuals were all examined during the same ten-day period of the Easter vacation. In with related factors and has emphasized the need for the continuation of studies on the fundamental factors in these various phenomena.

During the past year my effort has been continued on the analyses of both the mineral and the activator content of the various products, including the foods of both human beings and domestic animals. My researches on the seasonal tides and local levels of the activators in dairy products produced in different places at different periods of the year and for com-

parison of different places at the same time have been extended so that samples of cream, butter and cheese are now being received at the rate of about 700 per month from many countries throughout the world. It is of great importance that, in general, curves that are now produced for the first six months of 1930 show a remarkable similarity to those of the same months for the years 1928 and 1929. These are shown as general averages for a five-state area in Figure 2. It will be noted that the general levels were a little higher in 1929 than in 1928, and that for the first six months of 1930, they are higher than for either 1928 or 1929. This is doubtless due to the earlier spring growth caused by rains and warmer weather. They seem to have predicted not only an early summer but also an unusually hot one for 1930. Vitamin A is shown in the dotted line and vitamin D in

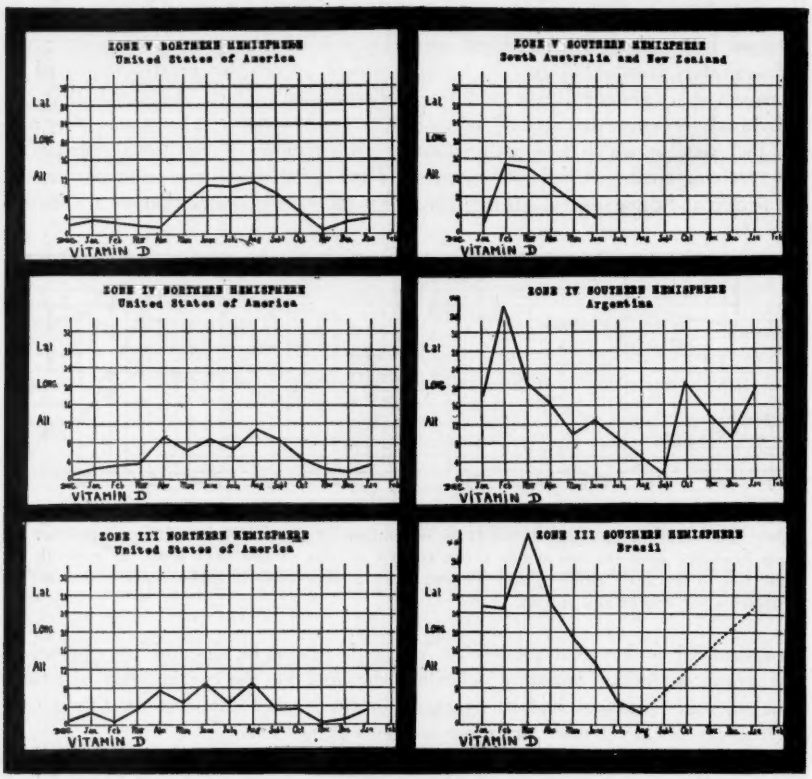

Fig. 3.—Comparison of northern and southern hemispheres by latitude zones for vitamin D in butters. An important similarity in the northern and southern hemispheres is shown to exist with regard to butter samples tested, except that the seasons are reversed. The remarkably good showing of some countries in the southern hemisphere is significant.

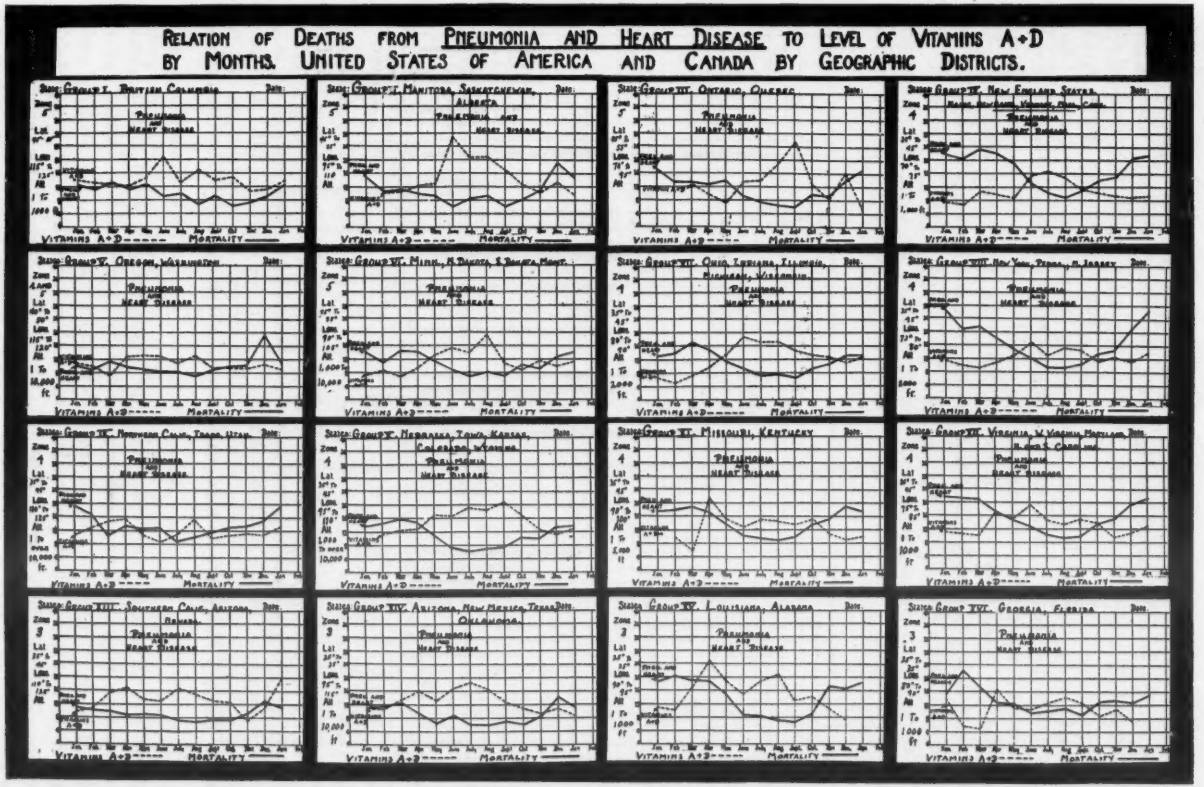
RELATION OF DEATHS FROM PNEUMONIA AND HEART DISEASE TO LEVEL OF VITAMINS A+D
BY MONTHS. UNITED STATES OF AMERICA AND CANADA BY GEOGRAPHIC DISTRICTS.
VITAMINS A+D
MORTALITY

Fig. 4.—Combined mortality curve for pneumonia and organic heart disease, shown to be, in general, the opposite of the vitamin level A plus D when the United States and Canada are divided into sixteen general districts.

the solid line. These data are for a five-state area in Zone IV between latitudes 35 to 45 north. In other recent reports, I have shown vitamin D in four factors or levels.

The area being covered by these intensive studies has been progressively enlarged until it now includes many nations throughout the world in both hemispheres, and it is exceedingly significant that now that data are available for comparing the northern and southern hemispheres with regard to vitamins in dairy products, the curves are in reverse with regard to their high and low periods just as should be expected on the basis of seasons and their effect on plant growth for providing food for dairy cattle. These data are shown in Figure 3. It is also of interest to note, when comparing the southern hemisphere with the northern, that the summer peaks in some of the countries in the southern hemisphere are higher than the summer peaks in the northern hemisphere. The causative factors are being intensively studied. It will be noted that, in the northern hemisphere, Zone III, which constitutes the southern tier of states, latitudes 25 to 35, north, and Zone IV, the next 10-degree period, including the center tier of states, are exceedingly similar in that they each carry a sharp rise in April, a fall in May, a rise in June, a fall in July and a rise in August, with a rapid decline in November. This is probably a direct indication of the influence of climate factors, chiefly temperature and

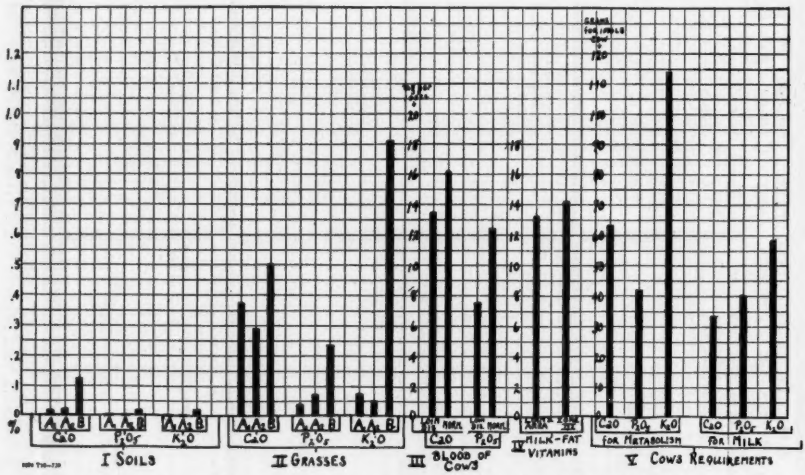

Fig. 5.—Study of a Texas cattle disease area on mineral and vitamin basis, considering grasses, cow's blood, milk-fat vitamins and cow's mineral requirements. In Groups I and II, A_1 and A_2, the composition of soils and grasses is associated with a loin disease area in Texas. *B* represents a knoll grass and its soil from Pennsylvania for comparison for calcium, phosphorus and potassium. In Group III, comparison is made of the calcium and the phosphorus of the blood of cows of the loin disease area. Group IV shows a comparison of milk-fat vitamins. Group V shows a cow's requirements for these three minerals for metabolism and for milk.

rainfall. The smoothness of the curves will be greatly increased when larger numbers of determinations are available, it being a physical impossibility as yet to obtain an adequate number of samples to represent these various districts properly.

difference in the levels of the vitamins in different places in the same latitudes at the same time. I have, accordingly, consolidated three types of data dealing with three entirely different phases of vital phenomena and I am presenting the in-

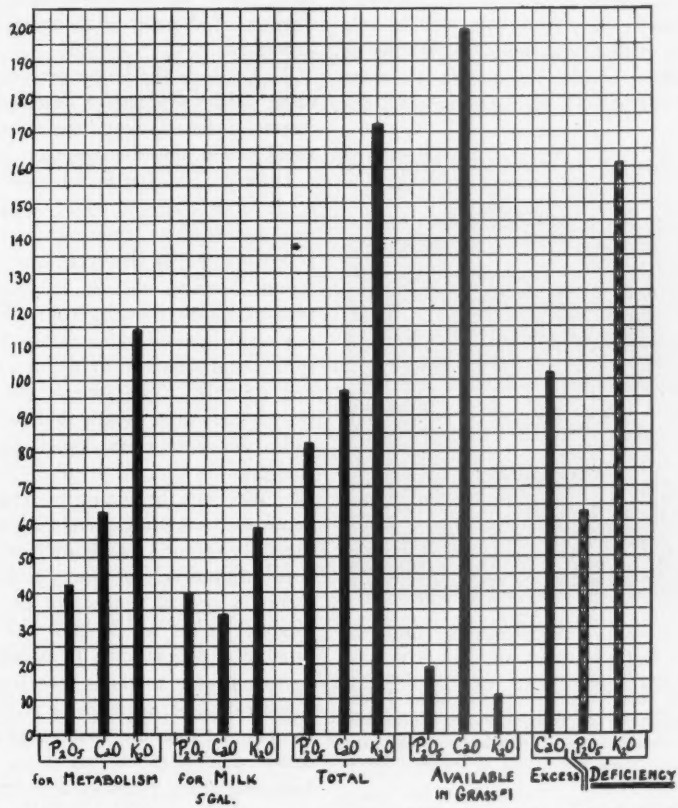

Fig. 6.—Mineral deficiency for 1,000-pound cow on No. 1 grass (loin disease area). This shows the metabolism and milk demands of a cow for minerals and the amount of these minerals available in a 56-pound daily ration of the Texas A1 grass. There is more than enough calcium, but a marked deficiency of both phosphorus and potassium.

Owing to the need for brevity, I cannot present data here in as complete detail as I would wish to demonstrate the great

formation in a form that makes comparison possible. I have divided the United States and Canada into sixteen different

districts each containing many thousand square miles and so related that they are comparable on the chart in their normal geographic relation to other districts. This presentation of these various phases is done in order that the coordinated problems may be studied under four headings, as follows:

1. To show the evidence of cycles in health and disease as illustrated by two of the typical and major affections of mankind; namely, pneumonia, an acute infectious process, and organic heart disease, a chronic degenerative process with contributing factors due to chronic and to recurring infection.

Fig. 7.—Above, animal making effort to rise after it had been down with loin disease for ten hours; below, animal suffering from loin disease, just found on prairie; some typical expressions of loin disease, which is primarily a mineral deficiency disturbance, with toxic poisoning from the eating of infected bones.

2. To present data dealing with variations in the total level of vitamins A and D in butter fats as determined in my studies from month to month throughout an annual cycle. These are determined from samples of butter and cream which I have obtained from individual creameries in various districts.

3. To study the possible relationship of cause and effect or common effects of the two factors, morbid conditions on the one hand and vitalizing forces on the other, in order to throw light on the etiology of diseases, both acute and degenerative.

4. To study the larger problem of the cause of the progressive development of disease, including dental caries, at a more rapid rate in some districts than in others.

It will immediately be seen from a study of the charts in Figure 4 that these diseases show a distinct cycle in their severity and that the cycle varies in different districts, though, in general, it tends to follow the same course and periodicity of increase in the fall, winter and spring and decrease in the later spring and summer. The general characteristics of this curve, which is shown in the solid line, is expressed as an increase in susceptibility or rate of deaths for these two affections, beginning in August and September and progressively increasing in most places until late winter or March, then progressively declining to mid-summer. A critical analysis of the various vitamin curves which show the sum of vitamin A plus vitamin D as determined by colorimetric methods reveals not only a general higher level of the vitamins for the summer than the winter, but also evidence of a sharp rise according to latitude, beginning probably at a time when the cattle are receiving rapidly growing young grass. In the southern latitudes, this rise comes in January and February, and, in many districts, it is followed by a later depression, which probably is related to precipitation affecting the vegetation. In general, the morbidity curve

is the reverse of the vitamin curve, and if it be true that plant life vitamins follow those of the cattle feeds as expressed in milk, there is strong evidence of a relationship of cause and effect through the development of acute infections and progressive tissue degeneration, with a reduction of the activators, some of which are known vitamins, and a marked decrease in these morbidity processes with a rise of plant vitamins. It is a striking fact that just as the vitamin rise comes later in the more northerly latitudes, the most rapid fall in the morbidities also appears later in the spring than in the more southern latitudes. There is also evidence of a summer depression in vitamins in several districts with the effect that the summer levels of these morbid conditions remain higher than in those districts with a high summer vitamin level. The conclusion seems logical, if not inevitable, that the life-giving factors as vitamins influence, if they do not determine, the level of the morbid conditions, and it seems most imperative to study the factors which determine the level of the activators in the food to ascertain why the available levels exist and also, if possible, to determine how they may be modified in order that the incidence of morbid conditions may be lessened.

The relationship that I have shown to exist between the degenerative diseases and the vitamin levels by months in the product of plant food, namely, milk, the only known complete food for life and growth, has strongly emphasized the need for research on the factors controlling the development of the activators or vitamins and also the levels of the minerals and other chemicals in the food products. It is of particular interest that many of the plant-eating animals of the world, if not most of them, are subject to periodic and often exceedingly severe disturbances which have been considered in the past to be mysterious diseases of unknown cause, but which have now

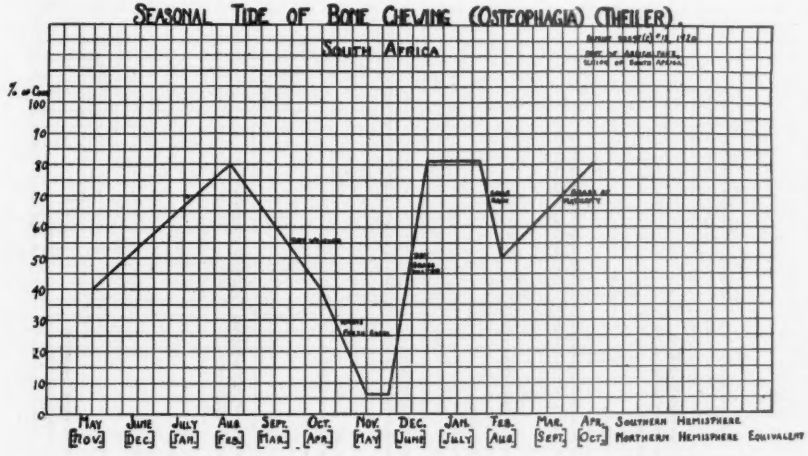

Fig. 8.—Seasonal tide in bone craving produced by mineral deficiency, which is a common and serious disturbance in some grazing animals in many countries.

come to be recognized as deficiency diseases, due chiefly to an inadequate supply of minerals and activators. This has resulted in the death of large numbers of domestic animals in various countries of the world at certain seasons and under certain climatic conditions.

My studies the last two years have included very extensive chemical analyses of soils and of plants growing upon them in relation to the health of animals and individuals living on those products. A kindness of Dr. H. Schmidt, veterinarian, acting for the chief at the Texas Agricultural Experiment Station (College Station), I have received samples of blood of affected animals for chemical analysis and also samples of the soils and two of the grasses. I will present here in detail results of six different types of determinations that have been made in a study of the causative factors or organ and tissue degeneration, due primarily to nutritional disturbance. In a more ex-

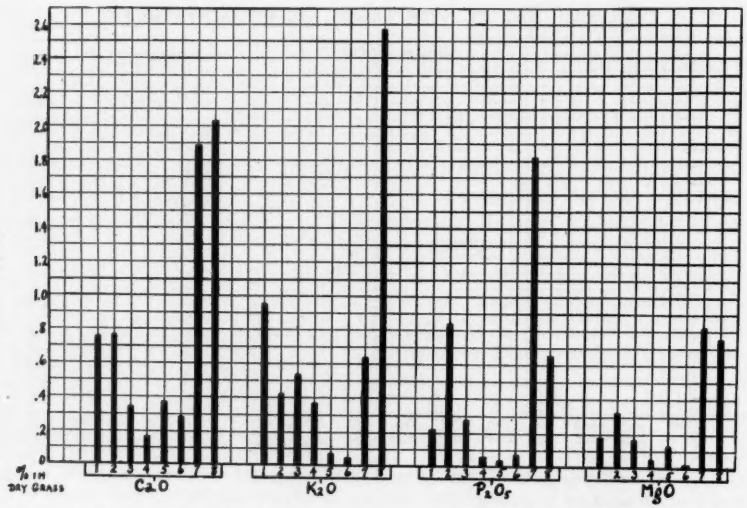

Fig. 9.—Comparison of mineral content of pasture grasses. *1,* Pennsylvania knoll grass. *2,* Ohio lawn grass. *3,* Arizona Johnson grass. *4,* Arizona crowfoot grass. *5,* Texas loin district grass No. 1. *6,* Texas loin district grass No. 2. *7,* Pennsylvania iron weed. *8,* British Columbia land kale plant. The difference in the chemical content of different pasturages from different places indicates an almost unbelievable difference in the mineral content that is possible and demonstrates readily the tragic stress that deficient grasses must produce in animals.

typical and striking illustration is shown in Figure 5, which represents an affection of cattle which appears in some of the coastal gulf counties of Texas and some other states. The cattle frequently go down with a disturbance locally known as loin disease. Through the tended communication, I am discussing the evidence of breakdown in one or more organs or tissues in domestic and wild animals in many countries throughout the world. While some of these, including loin disease, have an associated infection factor, the fundamental lesion

or disturbance is brought about by chemical deficiencies, chiefly mineral. Such disturbances in domestic animals are usually associated with an abnormal craving, particularly for bones, and treatment in many countries has as one, and usually the chief, factor, the feeding of ground sterile bone. Animals may be so afflicted with a craving for the minerals that they will eat even putrid bones and, by so doing, may obtain toxic material and bacterial infection from organisms such as *Bacillus botulinus,* and death may ensue. In many countries, members of this group of organisms are not present in the soil, and although the animals do not get this organism, large numbers die or are greatly reduced in efficiency. I have made these studies to throw light on human nutritional disturbance as well as that of the dairy cow. In Figure 5 I have presented the five following phases for study: (1) soils, the total and available plant food of calcium, phosphorus and potassium; (2) a comparison of two of the grasses (*A1* and *A2*) growing in the loin disease district with an average lawn grass taken from my front lawn (*B*); (3) the average calcium, phosphorus and potassium levels of the blood of three cows in the loin disease area and the average for fifteen normal cows; (4) the milk-fat vitamins for cows in the loin disease district and, in com-

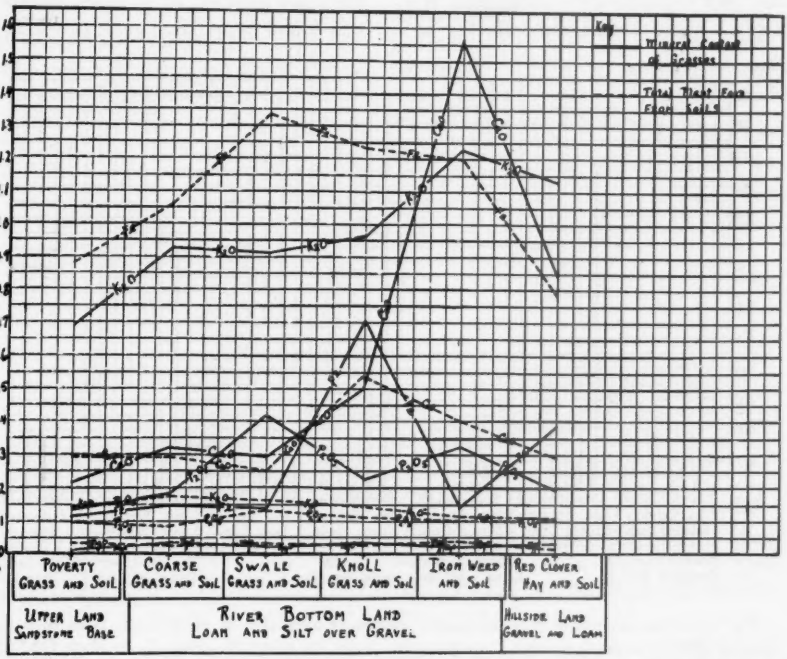
Key
Mineral Content of Grasses
Total Plant Food from Soils
%
Poverty Grass and Soil
Coarse Grass and Soil
Swale Grass and Soil
Knoll Grass and Soil
Iron Weed and Soil
Red Clover Hay and Soil
Upper Land Sandstone Base
River Bottom Land Loam and Silt over Gravel
Hillside Land Gravel and Loam

Fig. 10.—Curves showing mineral content of grasses and total plant food from soils from dairy district in northwestern Pennsylvania. The relation of soil poverty to grass poverty is directly revealed in the study of six grasses and the soils beneath them.

parison, my data for that entire latitude zone; (5) the daily requirements of a 1,000 pound cow, shown for, each, metabolism and milk.

The chemical analysis of the soils from beneath the grasses analyzed gives information bearing directly on factors controlling the quality of the grass. This is shown in Figure 5, Group I. The chemicals are shown in one form only, i.e., that available for each of three minerals. Most of the minerals are in a chemical form not available for plants. The calcium, phosphorus and potassium are all low in both Texas soils, the phosphorus and potassium being exceedingly low. In fact, with no other information, in the light of our present knowledge, we would expect that these two soils could not continue to maintain stock on the pasture they could grow. The levels shown here for available plant food phosphorus at 0.0007 and 0.0008 are only a fraction of that provided by a typically good soil, and the available potassium at 0.0004 and 0.0003 is likewise far below the minimum requirements for a good soil.

In Group II will be seen the chemical content of the grasses. The calcium of the grass marked *A1* is about one-third that found in my lawn grass B, which itself is low in calcium. Grass *A2* is about one-quarter that of my lawn grass. As we shall see later, even this low level of calcium is not the great embarrassment. The phosphorus contents of these two grasses are, respectively, 0.04 and 0.07 per cent of the dry grass, or one-twenty-eighth and one-sixteenth, respectively, the

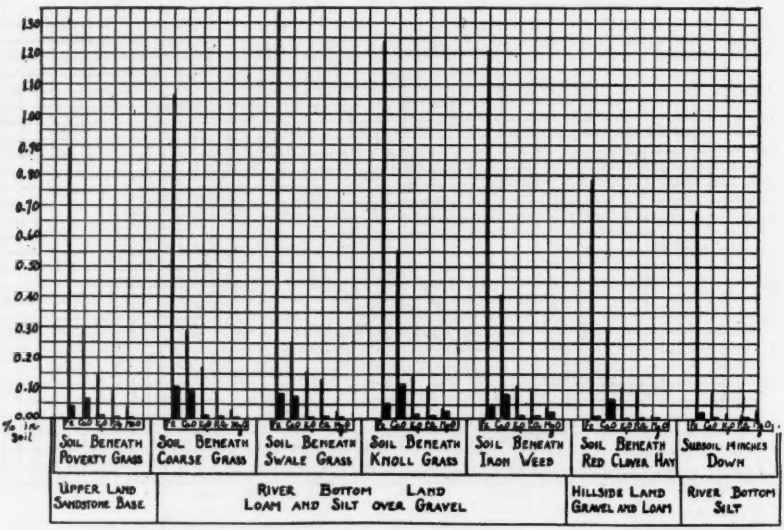

Fig. 11.—Comparison of total chemicals and those available as plant foods from soils from a dairy district in northwestern Pennsylvania. Since the minerals available as plant food must determine the limit of growth of plant life, it can readily be seen how depletion may rapidly destroy the fertility of the soil. Light line, total chemicals; heavy line, available chemicals.

quantity in my lawn grass. The potassium fares still worse. The quantity in grass *A1* is 0.07 per cent of the dry grass, and in *A2*, 0.05 per cent of the dry grass. That in my lawn grass is 0.49, seven times that in grass *A1*, and approximately ten times that in grass *A2*. We will see shortly the results that must be produced from the use of such fodder.

Since the blood of cows as well as the efficiency of the body will, of necessity, be dependent on the food elements available, we obtain important information from analysis of the blood for the level of various chemical elements, only three within the body. When disturbances appear in the mineral content of the blood or body tissues, they usually indicate a marked stress in which the body is not able to compensate by processes of readjustment and borrowing. The tissues, therefore, must suffer and function be impaired. Notwithstanding this remarkable compensatory capacity, blood sent to me for analysis of cows living on the pasturage of the loin disease area shows definite evidence of embarrassment. In Group IV, Figure 5, I have presented the levels of calcium, phosphorus and potassium in the blood of fifteen normal

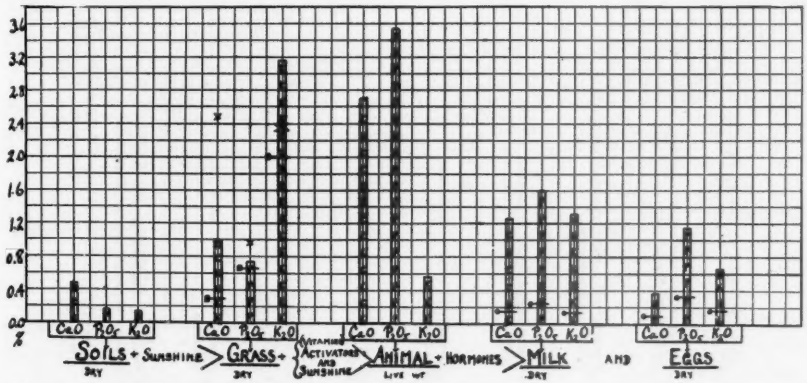

Fig. 12.—Comparison of mineral content of soil, plant, animal and food, x, extra good pasture; square, poor pasture; circle, wet weight. The route of the minerals from soils through grass to animal and to products, milk or eggs, indicates a stepping-up concentration from soil to animal.

of which are presented in this chart. In all of these studies, including soils, grasses, blood, milk, etc., a large number of chemical elements were determined which space does not permit of presenting here. It is of importance that the controlling mechanisms of the body are able to maintain relatively constant levels for most of the chemical constituents, owing to a remarkable system of temporary borrowing from depots of storage cows as controls and the average levels for three cows from the loin disease district. It is of importance that all of these three cows whose blood chemical content is shown here have had blood so seriously deficient in spite of the fact that each was receiving three ounces of bone meal per day, which, if it were all available, would go far to compensate for an ordinarily deficient pasture grass. The average calcium level in the serum of

normal cows in my series has been 11.5. The average for these three cows in the loin disease area is 9.7. The inorganic phosphorus of the blood of my series of normal cows has an average of 5.5 and in these three is 3.3. It has been known by the cattlemen of the affected districts that simply moving the affected cattle to another district for a few weeks or months would not only cure the disease, but also permit them to return to the same pasturage and frequently not break again for months. This demonstrates Nature's remarkable ability to replace depleted minerals in the normal storage depots of the body.

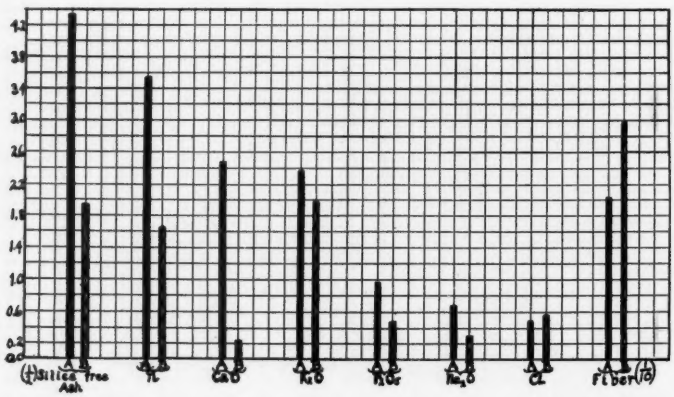

Fig. 13.—Comparison of pastures. *A*, Lord Ashton's race horse paddock in which superior stocks develop. *B*, Falkland Island pasture, where horses are reduced in size to ponies. The inevitable deterioration of animal life on deficient plant foods due to soil deterioration has been plainly demonstrated in horses transferred from English paddocks to countries with deficient soils.

An additional group of data are available from a study of the requirements of the body for metabolism and for milk production. This is shown in Group V, Figure 5. The daily requirements of a normal cow weighing 1,000 pounds for metabolism will be 42 gm. of phosphorus, 63 gm. of calcium and 114 gm. of potassium, estimated as their oxids. It has been estimated that a normal cow weighing 1,000 pounds can eat 56 pounds of fresh grass. The water content of the green grass making the difference between green and dry grass is usually estimated at 70 per cent. From the analysis of the grass shown in Group II, marked *A1*, which expresses the minerals in the percentage of dry grass, we see that that quantity of this grass would only furnish a total of 19 gm., which would be the limit of available phosphorus from that source for one day. We see by this that each day this cow would have to borrow from her storage depots, the tissues of her body, chiefly the skeleton, 23 gm. of phosphorus. Her requirements for calcium would be 63 gm. and the amount available in grass *A1* would be 199 gm. There would, therefore, be 136 gm. more than she would need for the daily requirements for metabolism. The needs of the body

for potassium are great. This 1,000 pound cow would require 114 gm. every day. Grass *A1* could only furnish her 11 gm. and her need would, of necessity, become very great. Were this cow being milked and producing 5 gallons of milk per day, which would not be a large amount for a good producer, she would require 40 gm. of phosphorus for that purpose in addition to that required for metabolism. Of calcium, she would require 34 gm. and of potassium 58 gm. for the milk alone. In other words, there would be a combined demand for both

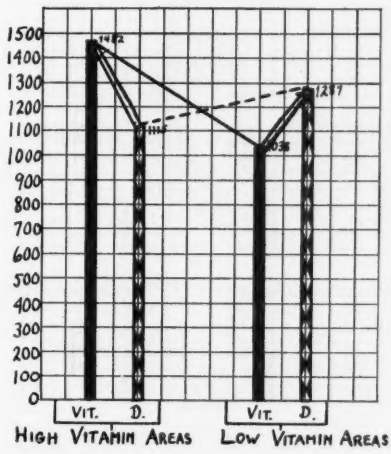

Fig. 14.—Relation of vitamins to deaths from pneumonia and organic heart disease. Sixteen districts of the United States and Canada are divided into two groups on the basis of vitamin levels. There is strong evidence that mortality levels are directly related to vitamin and activator levels.

metabolism and milk production of 82 gm. for phosphorus, 97 for calcium and 172 for potassium. These are all computed as their oxids. On a ration of grass *A1* alone, the daily overdraft for phosphorus would be 63 gm., and for potassium, 161 gm. The calcium demand would be satisfied with 102 gm. unused. About one-fifth of normal bone structure is phosphorus, one-third, calcium, and the remainder chiefly organic material. The 3 ounces of bone meal would provide only about 18 gm. of phosphorus even if all were utilized, which is probably never possible.

In Figure 6 is shown graphically this daily mineral shortage for phosphorus and potassium if only grass *A1* were used. In this chart, there are also shown graphically the mineral requirements of a 1,000 pound cow for, each, metabolism and milk production. There is no doubt that these cows found other grasses, though perhaps in limited quantity, to augment their potassium and phosphorus.

In Figure 7 will be seen two views of cows that are down with loin disease. Through the kindness of Dr. Schmidt, I am permitted to use these typical illustrations of this disturbance. Cows with loin disease at times go insane and attack those who are trying to help them. They often linger for days, dragging about their paralyzed parts in an effort to maintain nutrition. Somewhat similar deficiency diseases have created great havoc in many countries of the world. Expressions of deficiency diseases are generally a little different in one country from those in another, owing primarily to differences in the mineral deficiencies of the soil. In some countries, these conditions become so severe that when cattle and lambs are allowed to run together the cattle will kill and completely eat the lambs. Workers on biologic problems involving deficiency diets frequently find that if animals are placed together, one after another, members of the group in the cage will be missing, having been eaten by their associates. This, of course, requires that, in such tests, animals must be in separate cages.

The emphasis of recent years has been almost entirely on the rôle of infecting organisms without due consideration for either the mineral content of the food ingested and the mineral needs of the body or the rôle that these deficiencies play in establishing a condition of susceptibility to attack from infecting organisms. When we recognize that human beings are chemical machines like the cows and are dependent on the food intake for the maintenance of the structural units of the body, we realize at once that there is great possibility that imbalances will develop from inadequate diets. It is easy for us to place practically all the stress on the mineral intake together with the energy-producing food factors, with too little attention on the activators. While the activators include the known vitamins, they also include many substances not already identified or classified.

A most interesting incident occurred in connection with the cows whose blood we have just been studying with regard to loin disease in the coastal counties of Texas. According to Dr. Schmidt, within about a week, nearly all of the cows affected "went off bone meal." In other words, they suddenly lost their bone craving. This occurred with the rapid

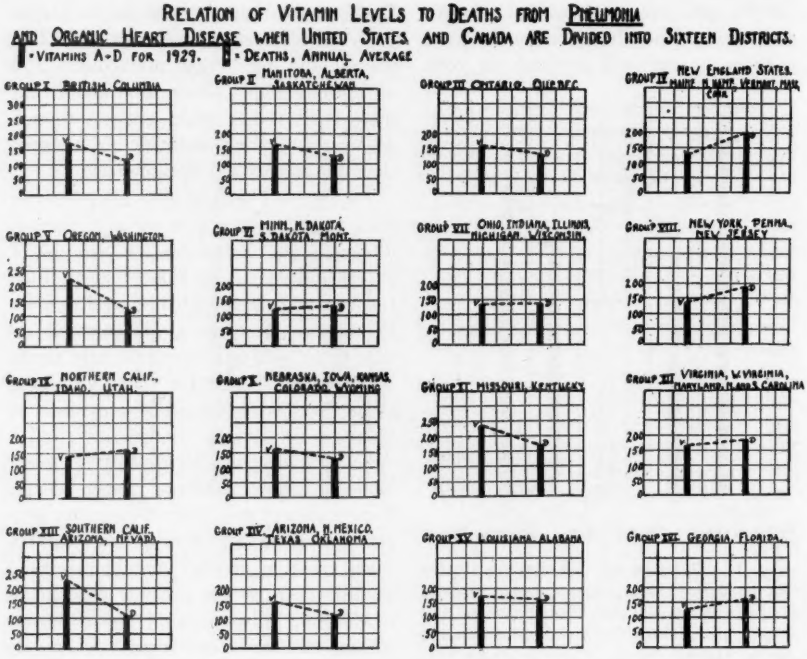
RELATION OF VITAMIN LEVELS TO DEATHS FROM PNEUMONIA
AND ORGANIC HEART DISEASE WHEN UNITED STATES AND CANADA ARE DIVIDED INTO SIXTEEN DISTRICTS.
V = VITAMINS A-D FOR 1929. D = DEATHS, ANNUAL AVERAGE
GROUP I BRITISH COLUMBIA
GROUP II MANITOBA, ALBERTA, SASKATCHEWAN
GROUP III ONTARIO, QUEBEC
GROUP IV NEW ENGLAND STATES. MAINE, N. HAMP., VERMONT, MASS., CONN.
GROUP V OREGON, WASHINGTON
GROUP VI MINN., N. DAKOTA, S. DAKOTA, MONT.
GROUP VII OHIO, INDIANA, ILLINOIS, MICHIGAN, WISCONSIN
GROUP VIII NEW YORK, PENNA., NEW JERSEY
GROUP IX NORTHERN CALIF., IDAHO, UTAH.
GROUP X NEBRASKA, IOWA, KANSAS, COLORADO, WYOMING
GROUP XI MISSOURI, KENTUCKY
GROUP XII VIRGINIA, W. VIRGINIA, MARYLAND, N. AND S. CAROLINA
GROUP XIII SOUTHERN CALIF., ARIZONA, NEVADA
GROUP XIV ARIZONA, N. MEXICO, TEXAS, OKLAHOMA
GROUP XV LOUISIANA, ALABAMA
GROUP XVI GEORGIA, FLORIDA.

Fig. 15.—Relationship of vitamins to mortality, shown to obtain in individual districts of the United States and Canada and suggesting a contributing factor through the progressive increase or mortality.

growth of the young grass and lasted for about six weeks, when a remarkable incident occurred. Within about four or five days, practically all of the animals again acquired the craving for bones. The usual method for testing for this condition (though often it will be immediately observable by the fact that the cows will eat pieces of wood, cloth or even dirt when bones are not available) is to place two grades of bones before the cattle to be tested and to note the percentage that try to eat them. Usually, two kinds of bone are used: some that are clean and white, free from all objectionable odors, and some putrid bones. A very marked craving is shown by the fact that they will eat not only the clean bones, but also the putrid bones. Why did the cattle acquire the craving so suddenly? Dr. Theiler, working in South Africa, where there has been a great deal of trouble with this affection, has presented data from which I have made the graph shown in Figure 7. By following the results outlined by Dr. Theiler as shown in the graph which I have developed from his data to more easily visualize them, it will be seen at once that the intensity of the hunger is not related directly to either sunshine or rain alone or even the two together. A note of caution

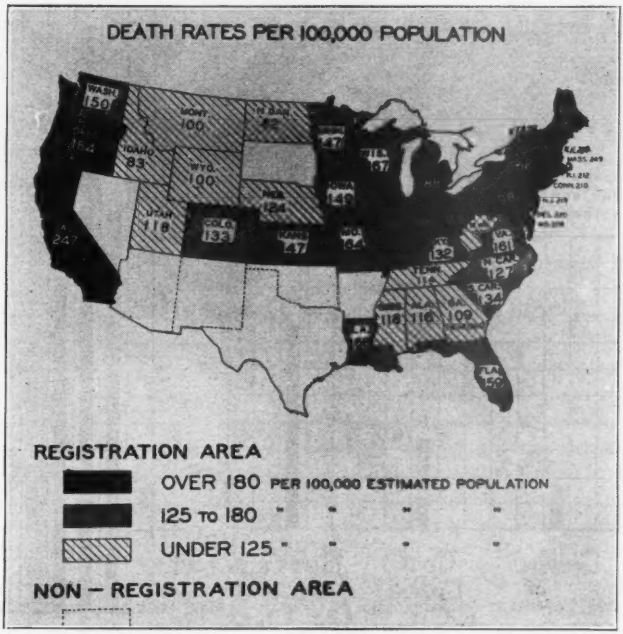
DEATH RATES PER 100,000 POPULATION
REGISTRATION AREA
OVER 180 PER 100,000 ESTIMATED POPULATION
125 TO 180 " " " "
UNDER 125 " " " "
NON – REGISTRATION AREA

Fig. 16.—Heart disease death rates, all forms, by states, United States registration area, 1925. The high mortality rate of certain areas of the United States are seen to have an important relationship to the vitamin levels and suggest a soil depletion by comparison with Figure 15.

is probably justified, in that individuals living in a particular latitude in either the northern or southern hemisphere will be in danger of thinking of seasons in terms of their expression in their geographic and physical location, whereas quite different factors will obtain with tude in which Dr. Theiler's work was done was approximately 40 south. During the period commencing with the beginning of the winter season, which is not a cold season in that latitude, and progressing through May to August (our November to February), there was a

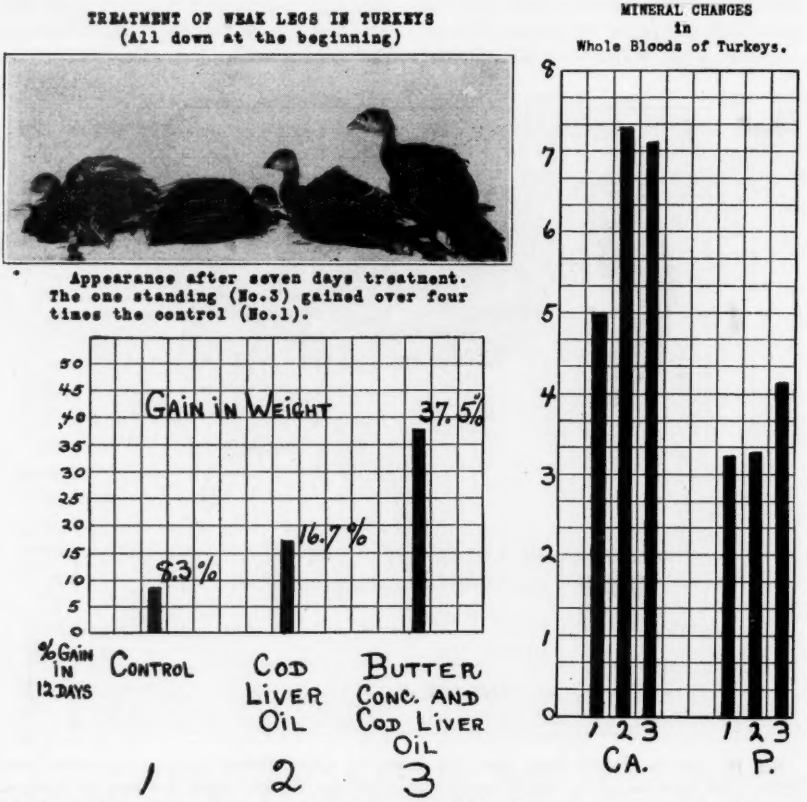

Fig. 17.—Treatment of turkeys with weak legs. Six turkeys were down with weak legs. The effect of treatment is shown to be greatly in favor of a mixture of the activators as concentrated from a high vitamin butter with a high vitamin cod-liver oil, over cod-liver oil alone. The turkey standing gained over four times that of the control and over twice that of the one on cod-liver oil.

regard to the period of growth as related to sunshine, maximum heat and cold periods, seasonal rainfall, etc. The lati- continual increase in bone chewing. Note that, at this point, 80 per cent of all the cattle ate rotten bones. From August 5

to October 27, there was a continual diminution of the curve, at which time it had fallen to 40 per cent. Note that during this period Dr. Theiler states: "The weather had been continuously dry, but as it grew warmer, the spring vegetation began to appear, and this is regarded as responsible for the diminution in pica (bone chewing)." At this time, rains began, lasting for a fortnight,

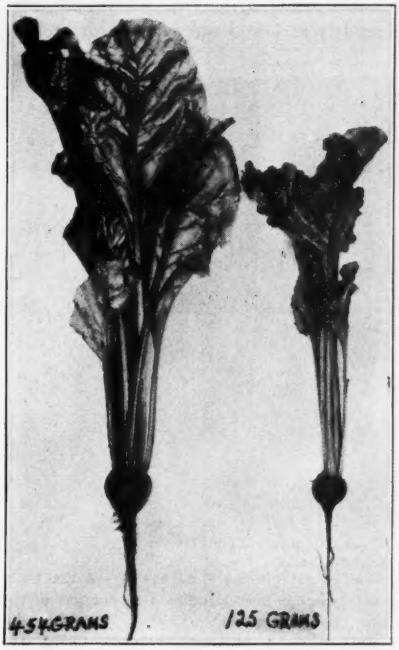

Fig. 18.—Remarkable difference in growth of beets produced by the addition of iron in small quantities to the soil.

with the result that "the young vegetation became almost luxuriant, and by the midd'e of November, the craving had rapidly fallen to 6 per cent." We see, then, an association so far with the rapidly growing grass. A number of important factors are recorded after a fortnight of rainless weather: the young grass wilted, and although the craving remained at the low level of 6 per cent for two weeks, in the next three weeks it increased to the high level of 82 per cent, the highest recorded, around which figure it oscillated for the next two months of summer drought. Clearly, something had happened to the grass so that, in its current state, it could not supply to the animals that which they needed so acutely to prevent the abnormal craving. As Dr. Theiler has emphasized, we have the record that the craving increased while the cattle were eating the old dry grass of the preceding year, prior to the beginning of the growth of new grass in August (their midwinter, equivalent to our February), and that again, during the period of dry weather when the grass was wilted in their summer, December and January (our June and July), the craving was acute. In the middle of January, "The drought broke, the pasture recovered, and it was naturally thought that the craving would again disappear. The unexpected happened, however, and notwithstanding the ample supply of good green grass and the absence of wilting, the craving did not fall to the previous 6 per cent, but only to 50 per cent." He states further that "by the end of March (our September, the end of their summer), the craving again stood at 80 per cent." It is of particular interest that Dr. Theiler relates these phenomena to the phosphorus content of the growing vegetation.

It is very evident, from many sources of information, that the utilization of minerals in foods is often directly dependent not only on a sufficient quantity of the particular minerals in available form, but also an adequate quantity of certain activators, some of which are

the known vitamins. For the cow, these must be available in the grass which she eats. If she can obtain sufficient of both the minerals and the activators for the needs of her own body, she will, without depletion of her skeleton, provide them for either lactation or gestation. Nature has provided that, in case of shortage, the mother's body will be depleted. This depletion may be offset and, under normal condition, the restoration is completely accomplished during the period of rest after stress. The nature of the activators, whether the known vitamins, including both the water-soluble and fat-soluble, or those regarding which little is known, would seem to be almost dependent on several factors; namely, the character of the plant, the soil on which it grows, the available moisture and temperature and the radiant energy, with regard to both the kind and quantity which nourishes and develops it.

We have naturally thought of grass such as cows eat as being a similar thing wherever found. It looks green and has many physical factors in common. In Figure 9 I have shown in groups for comparison grasses that I have studied from the chemical standpoint and for their vitamin content. These grasses selected are from eight different sources and have been studied for four of their minerals, namely, calcium, potassium, phosphorus and magnesium. They are numbered from 1 to 8 successively for each of the four chemicals. No. 1 is a Pennsylvania knoll grass which I obtained while searching for the reasons that high vitamins were being produced by a particular herd feeding on this farm. No 2 is an Ohio lawn grass growing on the front lawn at my office. Nos. 3 and 4, called Johnson and crowfoot grass, respectively, were from an Arizona district that showed evidence of nutritional dis-

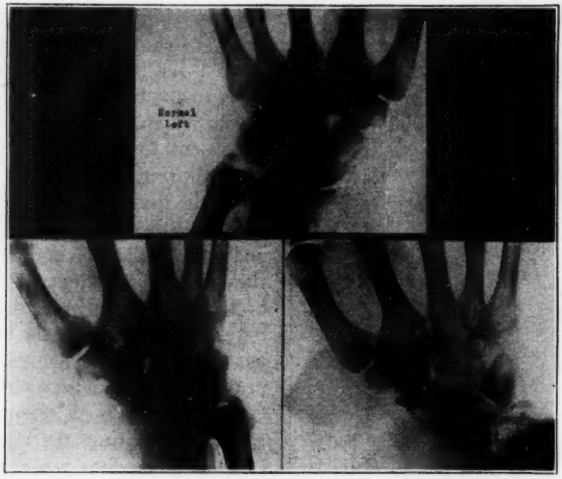

Fig. 19.—Changes in deforming arthritis with use of activators. The change in the right hand in a year in this case of multiple deforming arthritis has been produced by the use of the activators for assisting in the mineral metabolism.

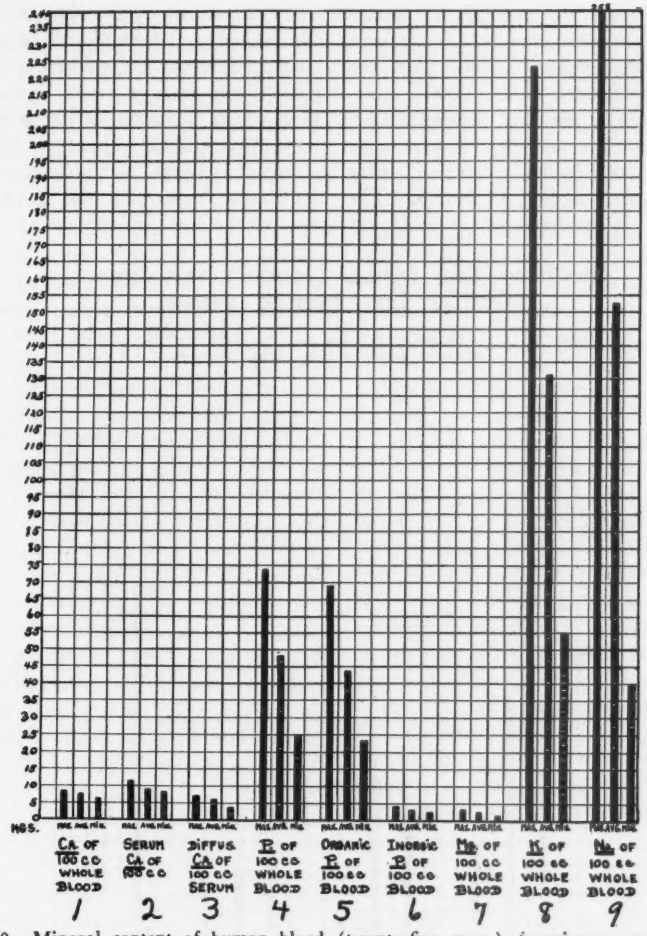

Fig. 20.—Mineral content of human blood (twenty-five cases) (maximum average and minimum levels). Notwithstanding the remarkable capacity of the blood building mechanisms to maintain standard levels for minerals, there are seen here quite wide variations chiefly as depressions though not so great as in plants.

turbances. Nos. 5 and 6 are the two Texas loin district grasses, the first of which we have just been studying. No. 7 is a plant that the cows were eating with great avidity on a farm in Pennsylvania. This plant is locally called iron weed. No. 8 is a pasturage shrub eaten liberally by the stock in British Columbia and called land kale. A glance at this chart reveals a remarkable difference in the quantity of the minerals in the grasses grown in different places. For example, the variation in calcium is from 0.17 per cent of the dry grass in the Arizona crow

foot grass, to 1.9 per cent in the Pennsylvania iron weed and to 2.0 per cent in the British Columbia land kale, the last two having ten times as much available calcium as the most deficient grass. The variation in potassium is seen to be very great, that in No. 8 being over fifty times as great as that in No. 6. The variations in phosphorus are also very great, that in the Texas loin district grass (No. 1) that we have just been studying being only one-forty-sixth that in the Pennsylvania iron weed. Similarly, the variations in magnesium is about sixty (?) times as great in the Pennsylvania iron weed, which was being eaten ravenously by the cows, as that in the grass No. 2 from Texas. From this chart, we see at once where we might go for some grass or fodder to re-enforce that which is found to be so deficient both from clinical experience and from chemical analyses. Similarly, I will present data illustrating the great difference in the chemical content of the same plant foods which we human beings eat when grown on different soils.

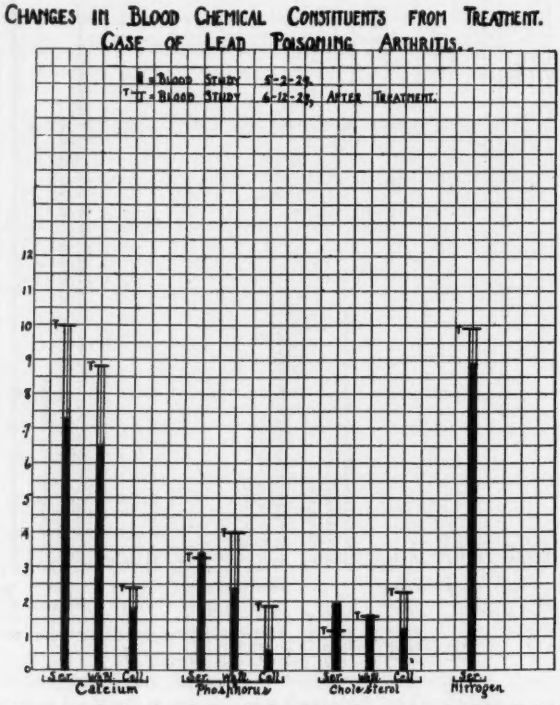

Fig. 21.—Improvement in levels of chemicals of blood associated with clinical improvement of last reported case of arthritis.

In the extensive studies that I have been conducting for several years on the vitamin content of dairy products as produced in different places at the same time and the same places at different times,

studies which now include about 700 samples per month of cream, butter and cheese, we are finding that there is not only a yearly cycle that tends to present in each individual district, but that the time of high vitamins corresponds with the period of rapid growth in young grass and that it is not readily produced by dry feeds and rations such as grain concentrates. I have studied a large number of grasses and cattle feeds in order to relate their chemical content, both mineral and organic, including vitamins, to their ability to produce a high vitamin milk product, and evidence is rapidly accumulating in large quantity indicating that the mineral and vitamin content of the foods constitute controlling factors for those of health with which we are directly concerned. Butter samples coming from districts that are characteristically high or low provide, by comparison, means for studying the controlling factors for vitamin levels.

The butter samples that have been received from a particular district in northwestern Pennsylvania were conspicuous, as I have indicated, by their high vitamin content, and I, accordingly, made trips to the district personally to study the water supply and other contributing factors and to obtain samples of the soil and grasses. I have already discussed only two of the grasses from this area; namely, knoll grass and iron weed. The physical characteristics of this district include a river valley about a mile in width, with sloping country on each side, the river being about 200 feet below the level of the surrounding country. The lowland is about from 6 to 12 feet above the summer level of the river, and the river bed is from 100 to 300 feet wide with a gravel bottom and only an occasional coarse boulder. This is shown in Figure 9 under river bottom land, the soil of which is loam and silt over gravel. The country opposite the river basin has a sandstone base and this is shown at the left in Figure 10. The hillside land is chiefly gravel and loam and this is at the right in this chart. The upland grass growing over the sandstone base of which specimens were taken is called poverty grass. From the river bottom land, I selected two grasses that the grazing cattle were not eating and a group of grasses and a plant that they were eating. On the hillside land samples of red clover were taken. The soil immediately under each type of grass was also taken for analysis, and the minerals available as plant food in the soil are shown in this chart in the broken lines, and the mineral content of the grasses growing upon these soils, in the solid lines. It is of interest that the cattle were wandering over the area apparently in search of the iron weed, which is shown by the graph to have a calcium level many times that of the first three grasses. The calcium was very high in the soil beneath the knoll grass, which had very short roots. It was also high under the iron weed. I am advised that the iron weed got its name from the size and strength of its roots, which penetrated very deeply into the gravel and therefore reached food sources at much greater depth than the surface grasses, which probably accounted for its high calcium level. I obtained additional samples of this plant in the early part of this season for comparison with the product of last season. The chemical analysis of this younger growth shows the calcium to be even higher than that tested last season. The calcium of the dried leaves of the plants which the cows were eating as obtained last year constituted 1.55 per cent of the total plant dry weight. That obtained this year from younger plants constituted 1.89 per

cent, which is very high. The phosphorus in the mature leaves of last year was 0.3 per cent, and in the more rapidly growing younger leaves of this year, 1.8 per cent, or six times as great. We see at once one of the characteristics of the rapidly growing young plant life, which is able to produce not only minerals for the cow, but the activators, including the vitamins, with which she will provide life-giving qualities for her offspring or her adopted wards. It is of interest to note that the phosphorus of this iron weed is 450 times that of the No. A1 grass in the Texas loin disease district. It is said that the roots of the iron weed have been known to penetrate four feet into the gravel, which would enable it to bring up from that depth minerals that would be completely out of reach of ordinary plants. It is also of interest to note that the potassium of this plant is nine times the quantity of that found in the Texas grass. We see at once the need to test the possibility of growing this iron weed plant on the Texas soil in order that it may, by the great penetrating power of its roots, bring up from the subsoil supplies of, each, calcium, phosphorus and potassium, so badly needed for the grazing stock of that district.

In this connection, it should be instructive to observe the marked difference in total mineral in the soils in question and that which is available as plant food. These determinations are shown graphically in Figure 11, from which it will be seen that only a small percentage of many of the minerals present in the soil are really available for plant food. The light lines indicate the total and the heavy lines total available chemicals. Plants are largely limited to water-soluble binary compounds.

In order to further visualize the route of the minerals from soil to human beings, and other animal needs, I have shown, in graphic form, in Figure 12 the relation of each to the other of soils, grasses, animals and animal products, namely, milk and eggs. The soils plus water and sunshine and germinating seeds will provide the needed minerals. Only three of the chemicals are shown in this chart: calcium, phosphorus and potassium. The grass plus the activators and vitamins which it carries, together with the action of radiant energy, as sunshine, nourishes the growing animal and stores these chemicals at much higher concentration. The grass is shown as dry weight and the animal as live and therefore, wet, weight. These data, since they were not found available in the literature, are obtained by analyzing an entire adult chicken, and they show the average chemical content of all tissues of the body. The animal, plus hormones and sunshine and utilization of the grass and its activators, is able to produce a highly concentrated animal food in the form of milk in the mammalia and as eggs in the egg laying species.

It has long been known that farm stock tends rapidly to deteriorate in size and efficiency as well as for milk production and reproduction on certain soils. It is common practice to grade up stock, whether dairy cattle, sheep, horses or pigs, by importing particularly favorable sires or dams and in this way improve the cross of the group or herd. It is also common knowledge that these efforts often result in a very temporary benefit. Too little emphasis has been placed on the importance of the plant food used as pasturage, which not only must provide the physical structures of the animal, but also must control its fundamental efficiency. As an approach to this problem, I am presenting, in Figure 13, a comparison of two pastures: *A*, Lord Ashton's

race horse paddock, on which superior stock develops, each generation being a little better than the one preceding, and *B,* Falkland Island pastures, on which horses such as those imported from Lord Ashton's race horse paddock are progressively reduced in size not only in their own generation, but rapidly in succeeding generations so that, in a few generations, they are reduced in size to that of ponies. The solid black line marked *A* shows the amount of the various chemicals in Lord Ashton's pastures, and the open work column, *B,* the amount in the Falkland Island pastures. The latter has less than half as much silica free ash as the former, less than one-half as much nitrogen, about one-tenth as much calcium, five-sixths as much potassium, about one-half as much phosphorus and about three-fifths as much sodium, but has a higher concentration of chlorin or acid factor and a much higher factor of fiber. I shall presently speak of this reduction in size of the body with deficient mineral and other chemical intake as it applies to human beings. In Figure 3, I presented data indicating the levels of vitamin A plus D, shown in the broken line, and the levels of the death rate from the two diseases, pneumonia and organic heart disease, in the solid line, and I have shown that, in general, one is the reverse of the other. It is of particular interest that this condition obtains not only for the entire territory of the United States and Canada, when considered as a unit, but also when that large area is divided up into sixteen smaller districts. It will also be seen, by referring to that chart again, that there is a marked difference in the average levels of these vitamin factors as determined from dairy products for different divisions of the larger territory. In Figure 14, it will be seen that, when we add together the monthly totals representing the vitamin units for each of these sixteen districts and then divide them into two groups of eight on the basis of these units, one of these groups contains 1,482 vitamin units as compared with 1,038 in the other, the former being approximately one-half greater than the latter. Similarly, I have determined from available statistical data the total number of deaths from pneumonia and organic heart disease in each of these two groups and find them to have two quite different levels. In the territory with the high vitamin content, the total deaths are represented here by the figure 1,115, and in the group with the low vitamin level, this increases to 1,287. We see at once evidence of a difference in the capacity of a district to furnish vitamin substance which appears to have a relationship to mortality that may readily be that of cause and effect.

In order to study this problem further, I have expressed in graphic form in Figure 15 these same factors for each of the sixteen districts. It will readily be seen that, when the vitamin content is high, the mortality is low, and when the mortality is low, the vitamin content is high. It will also be discernible on study that certain large districts are characterized by certain phases of this factor. For example, there is a tendency for the vitamin levels to be low in the Atlantic seaboard states and for the mortality rate to be high.

When we study these data in connection with those provided by the American Heart Association, we have what appears to be important new light on the progressive increase in mortality in certain sections of this country, particularly from organic heart disease. This is illustrated in Figure 16, which is taken from the report of the American Heart Associa-

tion. From this, it will be seen that the annual death rate from heart disease is very much higher in those states that are colored black, the figures reading 273 per hundred thousand of population in Vermont; 240 in Maine; 249 in Massachusetts; 252 in New Hampshire; 262 in New York; 82 in North Dakota; 100 in Wyoming, and 124 in Nebraska. In general, it will be seen to be lower throughout the middle western states. California and Oregon both show high rates, particularly the former, probably owing largely to a movement of people suffering from heart involvements from the highlands to the western lowlands. Since animal life is entirely dependent on plant food for its maintenance, it might readily be assumed that the minerals of the soils and the plants they produce constitute the chief or only controlling factors, if, of course, the energy-producing chemical factors, such as carbohydrates, fats and proteins, together with the atmospheric oxygen, constitute the requirements for life and body building. It has long been known by those studying intensively in the biologic field that the purer the synthetic food, the shorter the duration of life that could be maintained on it. From this has developed the great advance in the knowledge of life from an increased understanding of the activators, some of which are the vitamins.

Many illustrations might be used at this point, but I have used one immediately at hand, the studies on which are still in progress at this time. A flock of young turkeys belonging to a friend were in danger of serious loss through the development of weak legs. This occurred notwithstanding the fact that they were on a widely advertised baby chick ration which was supposed to contain not only all the mineral content needed, but also an adequate supply of cod-liver oil, presumably to provide vitamins. These turkeys had excellent care and were out in the sunshine and pasture all day, but notwithstanding this, they were going down. A half dozen of them were brought to me, and these are shown in Figure 17. Two were kept for controls, one of which died within a few days. The controls were continued on the same diet; namely, elaborately compounded chick food, and were given a grain mixture furnished by the same company. The other four received, in addition, the following treatment: No. 3 was given one 0 size capsule (0.6 gm.) of the preparation that I am now providing for patients in my experimental group. This mixture consists of a concentrate of the vitamins from an exceptionally high vitamin butter mixed with an exceptionally high vitamin cod-liver oil. Both are selected on the basis of chemical analysis. No. 4 had cod-liver oil activated five minutes in the sun rubbed on both legs, which required it to be kept separate from the others. No. 5 had one capsule of an activated cod-liver oil, and No. 6, 5 per cent of a high A and high D butter concentrate mixed with its regular food. Turkey No. 6 got its foot injured and observation of it was discontinued. Roentgenograms were made of one leg of each turkey. Space does not permit of presenting here as much detail as is desirable. The results are very clearly illustrated in Figure 17, which shows No. 3, the turkey on the activators obtained from a high vitamin butter and high vitamin cod-liver oil, to be in much better physical condition than any of the others. While one of its legs which was badly twisted and deformed when the treatment was started developed in an abnormal position, making it walk with a distinct limp, it is nearly normal and has outdistanced all of the others. I have

recently discussed in different communications the inability of vitamin D alone as activated ergosterol in any of its forms to completely correct this condition of weak legs in chickens. The percentage gain for this turkey (No. 3) was 37.5 per cent in twelve days, while the control on the standard chick foods gained 8.3 per cent. The turkey on the cod-liver oil plus standard diet gained 16.7 per cent. Blood chemical studies made on the thirteenth day are shown in Figure 17. The whole blood calcium for No. 1 is at 5 mg. per hundred cubic centimeters and in 2 and 3, both of those treated, over 7. The phosphorus is highest in No. 3. These changes are due to activators.

In several of my research reports, I have discussed the matter of physical deterioration with regard to the utilization of the stored minerals of the skeleton for meeting the daily needs of metabolism. I have referred to this as burning the furniture. One of its frequent expressions is the easy fracturing of bones, particularly of elderly people, though quite often of young people, and the difficulty of obtaining union. I have also presented data indicating the marked improvement in this regard through the use of the preparations built up to provide the activators in accordance with my views. In Figure 12, I have shown how horses that continued to grow larger and larger in succeeding generations on an exceptionally fine pasturage were reduced in size to ponies in a few generations on a very poor pasturage. This strongly suggests that nature adapts the size of the body, in large part, to the available food. No doubt, this has had much to do with the development of animals to enormous size in favorable periods of growth and similarly has controlled the size of the skeleton for many forms. It probably will be a surprise to many, as it was to me, to find that many of the patients who have come to me for study and guidance had already been reduced in stature. Cases of shortening of the body have been reported from time to time, some being quite extreme. One was reduced 12 inches in stature. One of my patients was found to have lost 4 inches in stature in five years, during which time she had been on a diet that had been prescribed to keep her weight down, literally one of starvation as to both minerals and vitamins. In less than four weeks, I have measured nine people who have shrunken in height an average of 1.6 inches within the last three to six years. One had shortened 2⅜ inches in height. All were breaking physically, with distinct evidence of degenerative breakdown which had been the reason for their having come in for study. A critical study of their diets indicated that these had been very incomplete both in minerals and in activators. In the last few years, I have had some cases of extreme shortening. I had warned the members of one family of the impending danger to their elderly mother who, according to her daughter's records had lost about 6 inches in height. One day, in walking across the floor, her hip collapsed. She, of course, became bedridden, later developed pneumonia and died from it in two weeks' time. I have a roentgenogram taken of her broken hip. The femur in size is that of a small child. Nature had endeavored to rebuild her body to adapt it to the limited material available, so much of it having been borrowed for the needs of metabolism. It is amazing how many people are burning their furniture at and before middle life. This has come about through two great stresses; namely, an inadequate supply of the building blocks needed for

the repair and maintenance of the body and the activators needed for enervating the tissues and for making the minerals available even when present in the food and also entering into tissue building. When we study the cause for breakdown in the tissues of the cows with loin disease, due primarily to a deficiency of the soil in certain minerals and the consequent deficiency of the pasturage thereon, we see at once means for reenforcing the ration of the cows to provide these emergency needs. An effort to do this was made and the cattle were fed bone meal, but it was only partially adequate. Incidentally, it would not furnish the potassium, which was shown to be so excessively low. From the table of grasses shown in Figure 9, it is easy to select additons for the ration of these cows that would reenforce at the point of weakening, it being assumed, of course, that the cows were dependent on grasses either *A1* or *A2*. This we would also do by improving the quality of the grasses available for pasturage, by improving the quality of the soil through mineral treatment, as part of a fertilization process. This is often not justified on account of the expense involved.

If space permitted, I would present here detailed data that I have obtained by analyzing for their mineral and other content samples of Marquis wheat grown from the same seed in twenty-one different places in Canada. The same seed developed marked differences when grown in different places. For example, there was over fifteen times as much iron in the wheat grown on the experimental plot of one section as on the experimental plot of one of the other sections. There are also wide variations in the calcium, phosphorus, potassium, sodium and other minerals.

The levels of the minerals in white bread as compared with those of whole wheat will vary considerably according to the wheat mineral content and the fineness of the bolting of the flour and also as to whether milk has been added. The calcium content of white flour is less than one-half that of whole wheat flour. The phosphorus and iron content are each about one-fifth that of whole wheat flour.

Since animals are dependent on plants to put the minerals into a chemical form that makes them readily available for utilization, and since, as we have seen from my data and the work of others, there may be a wide variation in the amount of minerals in a given plant, depending on the soil, we see at once a new approach to the problem of disease, particularly the degenerative diseases, including dental caries. Since the body is dependent on activators for efficient utilization of minerals in the food, even in the best plant food, and because the minerals are necessary for the maintenance as well as the growth of the body, both hard and soft tissues, we come at once to be limited by the amount of these minerals in given foods that may be available for utilization. When we study human morbidity problems as well as those of growth and health in the light of the intake of minerals and activators, we see ample reason for physical breakdown in one case, or, at one period, and normal health and function under other circumstances. We have at once the problem of making up the deficiencies by supplying foods from plant and animal origin that are capable of making good the shortages. Almost daily, I am studying people with regard to dental degeneration associated with degeneration of other parts of the body. We see by a study of their diet that Nature could

not possibly maintain body efficiency and that they could add to their dietary those supplies that would make a great improvement in their well-being. Probably no single group has impressed me more of late than that of boys and girls who have returned from higher educational institutions where they lived chiefly a dormitory life through the fall, winter and spring, and who showed an incidence of dental caries much above that of the general average of boys and girls of the same age period living under home conditions. One of my efforts is being conducted for the general betterment of this class. Some plant foods are particularly suited for providing high levels of certain minerals when grown on favorable soils. Lettuce, for example, has the capacity for carrying large quantities of iron. At the same time, it may contain little iron because of deficiencies in the soil. Tomatoes are probably our best food for providing copper, which is needed with the iron for blood building. Spinach is also a good carrier of iron, though not so good as lettuce. The quantity of available iron in the soil has an important influence in determining the amount of other metals that a given plant may take up. This is true of several minerals which, by the presence or absence of even a minute quantity to act as a catalyst, will greatly change plant growth. The influence of iron as a catalyst is well illustrated in Figure 18, in which will be seen two beets planted about the middle of May. About a month later, in accordance with my directions, there was placed in the surface of the ground and worked into the surface only a small quantity, about 1 ounce per square foot, of ferric ammonium citrate, one part of the row only being treated and the other part left without treatment. The difference in the growth of the two beets on the two different soils will be seen at once in this figure. When I photographed it, one would get the impression that there had been two different plantings, one later than the other. The gardner pulled some of both kinds to make a fair sample, and the largest beet in each group was selected for photographing and for making chemical analysis. The control, which is the smaller one, weighed 125 gm., beet and top, and the one from the treated soil 454 gm., an increase of 360 per cent. It is exceedingly interesting and significant that this enormous increase occurred without increasing the percentage concentration of calcium, phosphorus and potassium in the dry beet structure. The iron acting as a catalyst made it possible for this beet plant to build more vital structure without any increase in the amount of the other minerals in the soil. The amount of ferric ammonium citrate added was directed to be a tablespoonful for each square foot of surface without putting it close to the plant. This amount is about 1 part to 1,000 parts of the soil. This was done twice; first, at about five weeks after planting and again, ten days later, the same small quantity was added. There was, therefore, only three weeks' influence to produce the difference that obtained in the two parts of the row.

Another result that is obtainable from the use of special chemicals as plant food is changing of the amount of minerals in plants. Some lettuce that was treated in the same way shows 16 per cent more phosphorus in the lettuce growing in the soil that received the ferric ammonium citrate than that growing on the untreated soil. We have here means for greatly modifying the amount of minerals that may be available in a given article of food. It is very probable that this newer knowledge of the variations

in mineral content of given plants when grown on different soils will be utilized for the betterment of humanity. Various vegetable products will be sold on the basis of their chemical content. This may only require that we know the district in which a certain vegetable was grown for, in the light of my studies on wheat, there may be a wide variation of from 100 per cent to 1,500 per cent, depending on where it was grown and the mineral in question.

Unfortunately, space does not permit the presentation of as many clinical data as would be desirable for demonstrating the application of the new principles that have been suggested and emphasized in these studies. I will, accordingly, be able to present only one case as illustrating both clinical improvement and improvement in the chemical content of the blood as the result of carrying out the general procedures. The case is that of a very stubborn type of mineral utilization disturbance; namely, multiple proliferative arthritis in the hand. In Figure 19 will be seen one view of the left hand and two views of the right, the last two approximately one year apart, during which time this man had been taking additional activators in the form of a concentrate of the vitamins from a high vitamin butter mixed with an exceptionally high vitamin cod-liver oil, the dosage being two 0 size capsules (0.6 gm.) three times a day. It will be seen at once that whereas the carpal bones of the hand are fused together at the beginning of the treatment, they are distinctly separate and have an appearance quite near to that of the normal left hand after treatment. The stiffness of the hand completely disappeared, and the pain and swelling almost disappeared. Many other joints of the body had undergone a similar change. This patient had walked with difficulty and much pain, and he now stands erect and moves every joint with apparent ease and freedom. This man had been a painter and had previously had all of his teeth extracted. There was indication in the history of symptoms of lead poisoning. The relative and actual amounts of the minerals in human blood of twenty-five consecutive cases are shown in Figure 20 in three columns for each chemical: (1) the maximum, (2) the average and (3) the minimum. The averages are low normals.

The change in the blood chemical constituents in this case following treatment are shown in Figure 21. The solid dark portion shows the level at the beginning of treatment and the open column at T the level following treatment. It will be seen at once that the serum calcium, the whole blood calcium and the cell calcium have all increased. The phosphorus of the whole blood and cells increased. The cholesterol of the cells increased and decreased in the serum. With the taking of the activators, the patient was given direction relative to the emphasis to be made in the selection of his foods. Unfortunately, space does not permit of including a copy of the spectographic studies of the blood serum, urine and saliva before and after treatment.

SUMMARY AND CONCLUSIONS

1. Further data have been presented indicating that dental caries can be largely controlled by means of a program which provides an increase in activators and mineral intake.

2. Comparison has been made of the levels of vitamins A and D, as found in dairy products throughout various subdivisions of large areas, with the death rates from pneumonia and heart disease for the same areas. This indicates that,

in general, these two factors are in opposite phase; namely, when the recorded vitamin level is low, the recorded mortality rate for these diseases is high, and when the vitamin level is high, the mortality rate is low.

3. Evidence has been presented indicating that these forces do not have simply the relationship of being common effects of a common cause, but have evidently a relationship directly associated with those of cause and effect.

4. A detailed study has been presented on the relation of mineral and vitamin contents of foods to animal reactions in connection with cattle in a loin disease area and of the important influence of the vitamins alone in a study of turkeys. The data again indicate that not all of the needed factors are to be found in cod-liver oil.

5. Direct application of these new principles has been discussed and illustrated in a practical case showing the change in the levels of certain chemicals of the blood following treatment and associated with marked improvement in even so stubborn a disease as multiple proliferative arthritis.

6. Mineral deficiency in the soil from any cause may make it impossible for that soil to produce a plant life that is competent to maintain human needs, and this condition may be progressive with the continuous cropping and shipping away of the plant mineral concentrates. Data have been presented indicating that this is already an important contributing factor in the progressive degeneration of our civilization in those districts of the world, particularly demonstrated in the United States, which have been cropped most intensively for the longest period. This indicates that we are facing a problem of so great importance that it constitutes an actual challenge or threat to the endurance of the levels of human fitness. Fortunately, it also suggests important means for meeting the emergencies, which will include the prevention of dental caries rather than the repair of its ravages, and, similarly, the prevention of many degenerative processes of the tissues about the teeth and the long and serious group of sequelae following in the wake of the growth of bacteria in inviting mediums of the body competent to produce increased virulence and, therefore, ability to break down organs and tissues of the body.

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DISCUSSION

G. K. Dice, Tipton, Iowa: Will the removal of the calcifying teeth hasten the death of the individual?

Dr. Price: Do you mean will caries hasten the death of the individual? Carious teeth will become devitalized and furnish a focus of infection, and focal infection may attack that person at the time when the vitamins are low. You watch the funerals that go by your office. You will be surprised to see how many of those are heart cases. Just think of that in connection with this vitamin curve. By using additional vitamins, you will prevent not only infections that follow caries, but also the degenerative processes in other parts of the body which are likely to be going on at the same time as the caries. I might mention that reindeer milk is so rich in butterfat that it can be churned directly as it comes from the reindeer, and it has so much vitamin content that it constitutes practically a hundred times as high a level of vitamin per unit volume as the average cow's milk. For cows, the soil will determine the grass and the grass will determine the vitamin content of the milk.

R. M. Smith, Iowa Falls, Iowa: You spoke of cod-liver oil that one gets at drugstores as being very low in vitamin content?

Dr. Price: It is high enough in vitamin D to pass government requirements, but it is not nearly so high as I require for my use. If I had time to use the slides, I could show what serious injurious effects are experienced from giving large doses of cod-liver oil.

ANATOMY OF THE MOUTH AND ITS RELATION TO UPPER AND LOWER FULL DENTURE CONSTRUCTION*

By HAROLD LYTTON HARRIS, D.D.S., Minneapolis, Minn.

A COMPREHENSIVE and thorough knowledge of the anatomy of the mouth is essential if one is to keep pace with the progress that is being made today in prosthetic dentistry. When one stops to consider that the various technics that are before the profession today for the construction of upper and lower full dentures must necessarily have the same starting place, that is to say, an impression of the seating area of the denture, it becomes of paramount importance that the operator possess a thorough knowledge of the anatomy of the mouth, regardless of the technic he may use, if the denture that he builds is to be at all times in harmony with the anatomic structures involved in the seating area.

A survey which I have made of the literature of the past ten years on the subject of prosthetic dentistry in five of the leading dental magazines has shown that the greatest number of writers by far have been interested more particularly in articulation and occlusion than in any other part of the subject. Materials and their handling have been second in importance, and esthetics has been third; while the field in which we work, which is the basis of operation and the starting point for any technic, has been among the least to receive consideration from authors on this subject.

I appreciate that there is a divergence of opinion as to the relative value to the finished case of a thorough technic in impression taking, which obviously entails a thorough knowledge of the anatomy involved, and the articulation and occlusion of the case. There are some who hold the opinion that the articulating instrument to be used, and the balanced occlusion to be obtained, are of much more importance in assuring a satisfactory performance of the finished case than is the method of impression taking. This opinion I believe to be wrong; not that I believe the reverse to be true, but I do believe that the one step is as important as the other in relation to a satisfactory result in the finished case. The theory that a patient will more successfully wear a poor-fitting set of dentures having a balanced occlusion than he will a well-fitting set with malocclusion is perhaps acceptable, but I believe that we will have more successful cases when we pay as much attention to working in harmony with the anatomy of the seating area, as we do to working in harmony with the anatomy of the temporomandibular articulation.

There are certain fundamentals in upper and lower full denture construction which have come to be universally recognized. Russell W. Tench, of New York City, presented these in a most interesting

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and valuable paper at the Chicago Midwinter Clinic last January. Dr. Tench believes the consensus of opinion among leading prosthodontists to be that the peripheral borders of the dentures should rest on soft tissues, and that the operator should be skilled in a sound technic of impression taking to accomplish that result. In coordination with my discussion on anatomy, therefore, may I review for you a well-known method of impression technic which, in the opinion of many operators the country over, accomplishes for them the desired results.

The anatomy of the mouth and jaws, from the point of view of prosthesis, must necessarily be divided into three sections; namely, that part of the anatomy which deals with the foundation and border of the dentures; secondly, that which has more to do with the function of the dentures, the glenoid fossa with its surrounding anatomy and the muscles of mastication, and, thirdly, that which has to do with the esthetics of the dentures, or the muscles of expression.

It is the purpose of this paper to consider the first of these sections; namely, that part of the anatomy which concerns the underlying structures and the peripheral border of the upper and lower dentures, with the maxilla and the mandible, discussed according to (1) osteology; (2) muscles and ligaments; (3) blood vessels and nerves, and (4) mucosa, with a review of the anatomy of the structures forming the definite and specific border outline of the upper and lower dentures, which will be of interest to those who accept the theory that to obtain peripheral seal, it is necessary to build the periphery of the dentures in soft tissue.

First, a review of the difference in osseous structure of the maxilla and mandible would be tedious and probably uninteresting. Certain portions of each are important, i. e., wherein muscles attach, glands lie or blood vessels and nerves leave or enter.

The maxilla will be considered from the standpoint of its palatal anatomy and the tuberosity. The hard palate, which is formed by the junction of the maxillae along the median line, varies in shape in various mouths, from an inconspicuous formation, as seen in the mouth with the high vault, to a decided protuberance. In all cases, it is a nonstress bearing area as it is invariably covered only by a thin layer of mucosa. Posteriorly, it is formed by the spine of the palate bone, and, in certain cases, this spine lies posteriorly from the movable tissue; while, in others, it terminates anteriorly from the movable tissue from 2 to 6 mm. on the median line.

We may classify the upper portion of the mouth according to the width of the area between the distal border of the hard palate and the anterior border of the movable tissues of the soft palate, or in other words, throat form: Class 1, those cases in which there is a distance of 5 mm., or more, between these two points; and the throat form consists of a gentle curve from before backward; Class 3, those cases in which the hard area continued backward and the curtain of soft tissue dropped downward abruptly from it; Class 2, those intermediate cases offering a fair amount of area capable of withstanding the postdam pressure. In Class 3 cases, the operator, in building the posterior plate line, is faced with two alternatives, both of which are unfavorable. He may either stop the denture anteriorly, so that it rests on hard tissue, or he may extend it distally so that it covers a small amount of movable soft tissue. It is decidedly better to choose the lesser of these two evils, that is, extend the border of the denture distally and the postdam

on soft tissue, with the hope that the patient will be able to tolerate the tickling sensation that is usually caused by so doing. Stopping the denture on a hard area will result only in atrophy of the underlying tissue and a loss of denture seal, which will cause the operator no end of trouble.

The tuberosity is the inferior angle of the maxilla, articulated with the tuberosity of the palate bone. Its size and shape is effected to a great extent by the life history of the third molar and the manner in which it consequently was removed. The tuberosity high on its posterior surface gives rise to a few fibers of both the internal and external pterygoid muscles.

An osseous structure, not a part of the maxilla but in close relation to the tuberosity, greatly concerns the operator in denture work. It is the hamular process of the medial pterygoid plate of the sphenoid bone. It lies posteriorly and medially from the tuberosity forming a triangular area between these two osseous structures in which there is a resilient pad of soft tissue, the only area in that portion of the mouth capable of withstanding the pressure exerted by the postdam. The hamulus serves to deflect the bursa or tendon of the tensor veli palatini, and the tip of the hamulus gives insertion to the pterygomandibular raphe.

The hamular process is also a landmark for the greater or posterior palatine foramen, which lies anteriorly from it on the palatal portion of the maxilla. This foramen transmits the greater palatine branch of the descending palatine artery, along with the anterior palatine nerve.

The incisive, or anterior palatine, canal lies in the median line posteriorly from the incisor teeth. It is formed by the junction of the two maxillae, and consists of a large opening at the bottom of which are four foramina, two smaller ones, the foramina of Scarpa, being located directly on the median line. They serve to transmit the nasopalatine nerves, the left passing through the anterior and the right passing through the posterior. The other two are somewhat larger, and lie laterally from the median line, these being the foramina of Stenson, transmitting terminal branches of the descending palatine artery.

To apply the postdam pressure accurately, as will be recognized, the operator must make a careful study of this area from the tuberosity on one side across the soft palate to the tuberosity on the other side. The pressure exerted must vary with the depth and resiliency of the soft tissue which is to withstand the pressure. To accomplish this accurately, it is necessary to have an impression of that area with the tissues completely at rest, under no pressure whatsoever. For the case, an upper tray is selected that is from one-eighth to one-quarter inch over-size on the labial and buccal aspects and long enough distally to include the area of the hamular process. Compound is softened, adapted in the tray, inserted in the mouth and pressed to place with a firm pressure. It is allowed to cool, and then removed from the mouth. The compound is chilled thoroughly. The surface is then heated by gentle painting with a small flame, tempered in water of about 110 degrees and reseated in the mouth under firm pressure, tissue detail and markings being thus brought out. The excess compound is trimmed back to the tray, the tray removed and the compound further cut back to the height of convexity of the buccal and labial folds.

From this point on, frequent chilling of the compound in icewater, precluding any possibility of warpage, is essential. The compound tray is now ready for

muscle trimming. The buccal flange, from tuberosity to cuspid, is gently heated with a small flame at the periphery on one side at a time, tempered in warm water and inserted in the mouth under pressure, the patient's cheek being drawn down at the same time with the other hand. The flange is then removed and chilled and the excess appearing on the buccal is trimmed away. This process is repeated until no more excess appears to be folded over by the tissues. The opposite side is now done in the same manner, also the labial from cuspid to cuspid, the lip being pulled downward, side to side, and folded lingually over the process to insure accurate adaptation of the labial flange. In the muscle-trimming, it will be noted that the border tissues have been pulled beyond their normal functional range. The space making up the difference between the established range and the functional range will be taken up and accurately established by the plaster wash.

To achieve an impression of the area to be postdammed with the tissues under no pressure whatsoever, the compound over the hard area to be relieved and across the postdam area is reduced by from one-sixteenth to one-eighth inch in thickness, only the horseshoe-shaped stress-bearing area and the muscle trim being left intact. A thin mix of quick setting plaster is poured into the compound impression, replaced in the mouth and forced to place by a pressure of about 25 pounds exerted at a 45 degree angle to the occlusal plane. When the plaster has set thoroughly and the impression has been removed from the mouth, the compound over the alveolar area should show through the plaster completely, and there should be a heavy plaster impression 2 or 3 mm. thick of the hard area to be relieved and the entire posterior border of the impression covering the area to be postdammed.

By the use of an egg-shaped burnisher, or a similar instrument, the exact width and length of the hard area is determined and charted with an indelible pencil on the tissue, extending always far enough anteriorly to include the anterior palatine foramen. Then, with a mouth mirror, the notch between the hamular process and the tuberosity is located and marked with the indelible pencil. The posterior margin of the plate on the median line is then noted by having the patient say "ah" and placing an indelible mark 1 mm. anteriorly from the movable tissue, and the plate line is completed by connecting the tuberosity markings with a gentle curve through the median line marking.

The impression is then replaced in the mouth in order that the palatal markings may be transferred to the plaster. This marks the impression accurately as to the location of the hard and postdam areas. In those cases in which there is a decided undercut in the region of the tuberosities, these areas should be studied, and the palatal markings placed before the plaster wash impression is taken. The hard area is relieved by scraping away 1 or 2 mm. of plaster over that area, the amount depending on the prominence of the area. By the use of a sharp pointed instrument, the posterior plate line is grooved in the middle of the indelible marking, so that an embossed line will appear on the cast. The cast is poured, and the bite plate constructed; and when the patient returns, the case is ready for postdamming.

The postdam area is divided into five smaller sections of equal size. Starting on the left where the buccal and posterior plate margins meet, passing posteriorly around the tuberosity between it and the hamular process is Area 1. Medially from the process to a point 5 mm. from the median line is Area 2, and Area 3 is

the median line area, consisting of about 10 mm., that is, 5 mm. on either side of the median line. Areas 4 and 5 are similar to Areas 2 and 1, respectively. Now, with an egg-shaped burnisher, the tissue in Area 1 between the tuberosity and hamular process is completely compressed. The distance the burnisher traveled to compress this tissue is judged as carefully as possible. Two-thirds of the distance is computed, and is transferred to the cast by the use of a No. 10 round bur in the straight handpiece. A No. 10 bur is used because it is the width of the narrowest part of the postdam, the width of the area between the tuberosity and the hamular process.

A groove is cut in the cast along the embossed line indicating the posterior plate line in Area 1 to the depth as computed. Next, the amount of compression in Area 3, or median line area, is computed, which is obviously the least compressible of the five areas, and transferred to the cast. Areas 1 and 3 are then connected by the groove through Area 2, the depth of which has been computed as in Areas 1 and 3, and which is usually greater than in either of those two areas. Areas 5 and 4 are then handled in the same way, the result being a narrow groove across the posterior plate line varying in depth according to the tissues in the mouth which are to bear the pressure thus exerted. The next step is to widen the postdam according to the structure of the soft tissue of the palate.

In Areas 1 and 5, the postdam, as stated before, is never wider than the groove made by a No. 10 round bur, for the reason that the distance between the tuberosity and hamulus is constant, with only minute variations; therefore, merely the sharp edges of the groove are rounded over, a sharp knife being used for this purpose. Widening the postdam at this point would result in soreness on the tuberosity and atrophy to the underlying tissues. Posteriorly, it would result in an inflammation of the bursa of the tensor veli palatini, as it is deflected around the hamulus. In Areas 2 and 4, soft tissue is found anteriorly beyond the posterior palatine foramen. This pad of soft tissue normally is thicker by far than any other area of the postdam, and therefore is capable of being and should be postdammed. By the use of a sharp knife, this triangular area of soft tissue is scraped from the deepest part of the groove anteriorly to a feather edge, the depth of the postdam as it passes over the posterior palatine foramen thus being lessened.

In Area 3, or the median line area, the width of the postdam area is exactly that of the distance between the posterior plate line and the distal border of the hard palate, and is postdammed as before with a knife, the plaster being scraped anteriorly to a feather edge at the border of the hard tissue. When this is completed, the posterior portion of the biteplate is warmed and molded into the postdam depression of the cast. Postdamming with compound on the impression will not, in the first place, allow the operator to work with the tissues at rest, and, secondly will not consequently allow an accurate and definite computation of the width and depth of the postdam.

The muscles involved in the maxillary area are (1) the buccinator; (2) the levator veli palatini, and (3) the tensor veli palatini. The buccinator originates from three areas: the buccal aspect of the molar portion of the maxilla, the corresponding area on the mandible and the pterygomandibular raphe, and is supplied by the buccal branch of the facial nerve. Its fibers converge and are attached in the group of muscle fibers known as the orbicularis oris at the angle of the mouth.

The levator veli palatini arises partly from the inferior surface of the petrous portion of the temporal bone and partly from the lower portion of the cartilage forming the eustachian tube. It is attached in the aponeurosis of the soft palate. The nerve supply is from the pharyngeal branch of the vagus. The tensor veli palatini originates from the scaphoid fossa of the sphenoid, with some fibers from the lateral side of the cartilage of the eustachian tube. It passes forward, and is deflected by the hamular process, passing medially to be attached along the spine of the palate bone and partly in the aponeurosis of the soft palate. Its nerve supply is from the mandibular branch of the trigeminal through the otic ganglion.

The mucosa overlying the abovementioned portions and structures varies in thickness and therefore in resiliency. The thickness of the mucosa over the hard palate varies directly with the prominence of that area; that is, the greater the prominence of the hard area, the thinner the mucosa. It is strictly a nonstress bearing area, and must be relieved in every case. The mucosa overlying the alveolar crest, which is normally from 5 to 7 mm. thick, is capable of withstanding at all points the masticating stress of the upper denture. The mucosa overlying the posterior palatine foramen in five specimens averaged 8 mm. in thickness, postmortem measurement, and it is known to be invariably thicker than any other overlying mucosa on the maxilla. As the nerve and blood vessels emanating from this foramen run anteriorly in a groove, this area is capable of withstanding a slight amount of postdam pressure, rather than necessitating relief.

The incisive foramen area is thinly covered, and therefore must always be relieved. The mucosa between the tuberosity and the hamular process has been described previously and is capable of withstanding considerable postdam pressure. Near the posterior margin of the hard palate, there is, on each side of the median line, a small pit, a familiar landmark for determining the posterior border of the denture. These pits, the foveola palatinae, usually mark the beginning of the movable tissue of the soft palate.

The mandible, from the standpoint of peripheral anatomy of the lower denture, presents many sites of interest. Admittedly the largest and strongest bone of the face, the edentulous mandible varies in size and shape according to the history and condition of the natural teeth during their retention and their investing tissues, and, of course, according to age. It presents three surfaces in the areas in which we are at present particularly interested; namely, the lateral, medial and alveolar surfaces. The lateral surface presents, near the median line, or symphysis, a depression known as the incisive fossa in which arise the mentalis and incisivus labii inferioris muscles. Under the second bicuspid area is the mental foramen, from which emanate the mental nerve and blood vessels. The external oblique line arises from the mental tubercle, passes upward and backward below the mental foramen, passes medially to the attachment of the buccinator muscle in the region of the molars and continues on up the ramus helping to form part of the retromolar triangle.

The medial surface presents near the symphysis four small tubercles, which collectively are called genial tubercles, or the mental spine, and mark the origin of the genioglossus and geniohyoid muscles. Beginning just below the mental spine, and extending upward and backward, the mylohyoid line reaches its greatest prominence in the region of the distal cusp of

the second artificial molar, then disappears on up the ramus. The mylohyoid line serves as the point of attachment along its entire length from the mental spine to a point directly beneath the distal cusp of the second artificial molar for the mylohyoid muscle. From the distal margin of the mylohyoid muscle, the mylohyoid line is void of tissue attachments for approximately 5 mm.; and then, in what is the posterior fifth of the line, is attached the mylopharyngeal, or third head, of the superior constrictor muscle of the pharynx. Above the mylohyoid line beginning at its anterior border and extending to the distal aspect of the first bicuspid area is a depression on the mandible known as the fovea sublingualis, or fossa for the sublingual gland. Below the mylohyoid line in the molar area is another depression for the submaxillary gland.

The buccinator muscle, which forms the periphery of the molar area of the lower denture buccally, has its broad attachment directly inferior to the external oblique line, the fibers running upward and backward to join with the maxillary and raphe attachments. It, therefore, moves approximately at right angles to the denture, any overextension of which will tend to cause the buccinator to lift the denture from the ridge. Stopping the denture short of the attachment allows no seal for the periphery as the mandible at the point of the external oblique line is covered only with a thin layer of mucosa. Auxiliary buccinator attachments frequently occur in the regions of the bicuspid.

No other muscle comes in contact with the buccal or labial peripheral area, but as the distal border of the denture crosses the crest of the ridge, it passes over a pad of soft tissue, an area known as the retromolar triangle, the area lying distally from the last molar formed by the external and internal oblique lines. In this area are attached a few terminal fibers of the temporal muscle, and these fibers, with the overlying mucosa, furnish a soft tissue sealing area resilient enough to withstand a slight postdam pressure.

The mylohyoid muscle, referred to before, arises along the anterior four-fifths of the mylohyoid line, its fibers passing downward and inward forming a convexity which is the floor of the mouth. The muscle is attached to the ventral surface of the hyoid bone, and its nerve supply is from the mylohyoid nerve, a branch of the masticator, or portia minor. It also functions at right angles to the border of the denture from a point beneath the second bicuspid to a point beneath the distal cusp of the second artificial molar. Any overextension of the denture border along this line causes the denture to be lifted from its seating area by the action of the tongue, because of the complete coordination of action between the mylohyoid and geniohyoglossus muscles. Any underextension would result in loss of seal, as the periosteum is covered only by a thin layer of mucosa.

The mylopharyngeus muscle is involved in the distolingual peripheral area of the lower denture, that area known as the retromolar or lateral pressure area. The mylopharyngeus, in reality one of the three divisions of the superior constrictor muscle, derives its name from its origin on the posterior fifth of the mylohyoid line approximately 5 mm. from the distal end of the origin of the mylohyoid muscle, and from its insertion in the great fibrous raphe of the pharynx. The other two divisions of the superior constrictor are the buccopharyngeus, or that part arising from the pterygomandibular raphe, and the pterygopharyngeus, or that part arising from the hamular process of

the pterygoid plate. This group of muscles, with the middle and inferior constrictor of the pharynx, all have their nerve supply from the pharyngeal plexus. The action of the mylopharyngeus is parallel to the surface of the overlying denture, and therefore does not tend at any time to dislodge the denture from its seating area, merely forming a soft tissue area for obtaining peripheral seal.

The pterygomandibular raphe is a fibrous band of tissue running from the tip of the hamular process to the internal oblique line of the retromolar triangle on the mandible. It serves to give origin to the middle or second division of the buccinator muscle running anteriorly; and, posteriorly, it gives rise to the buccopharyngeus, or second division, of the superior constrictor, which furnishes, at its attachment, a soft tissue sealing area at the point where the border of the denture crosses the internal oblique line of the retromolar triangle.

The classification of throat form in the lower mouth is based upon the action of the palatoglossus muscle, and its overlying tissue attachment to the mandible. The palatoglossus is a cylindrical band of muscle having its origin on the oral surface of the palatal aponeurosis of the soft palate, and its insertion in the superficial muscle layer on the side and under surface of the tongue. Between its origin and insertion, it forms the anterior pillar of the fauces or tonsillar crypt. The muscle moves forward upon the extension of the tongue, carrying with it in a sweeping action its overlying mucosa, which is attached to the mandible. In a Class 1 case, then, there is a large amount of lateral pressure area left distally from the mylohyoid line when the tongue is extended to a point one-eighth inch below the red line of the lower lip. This lateral pressure area, which has had such misnomers as "the pretonsillar area," "flange area," or "undercut area," is practically obliterated in a Class 3 case, as the curtain of overlying tissue comes forward almost to the mylohyoid line on the extrusion of the tongue the distance given before. Class 2 cases, obviously, are intermediate of these two extremes.

The sublingual gland with its overlying mucosa comes in direct contact with the lingual margin of the denture from the frenum to the second bicuspid area. Its medial surface lies in a crypt made up of the mylohyoid, geniohyoid and genioglossus muscles; and when those muscles, together with the floor of the mouth, rise in the act of deglutition or in the act of extrusion of the tongue, the gland rises upward against the denture. Its nerve supply is derived from the sublingual branch of the lingual nerve.

A most interesting situation lies in the proximity of the lingual nerve to the border of the lower denture, necessitating, in some patients, specific relief at that point. This large nerve trunk, the foremost of the branches of the mandibular in position, passes medially from the inferior alveolar nerve, descending on the medial side of the external pterygoid muscle, where it is joined by the chorda tympani, a branch of the glossopalatine nerve which furnishes the sensory fibers to the tongue. It then passes between the internal pterygoid muscle and the ramus, continuing on over the fibers of the mylopharyngeus, passing forward behind the last molar area just above the mylohyoid line at its most prominent point. At this point, the lingual nerve is covered by the thin mucous membrane overlying the periosteum. The nerve then dips downward and forward toward the tip of the tongue, touching the lateral surface of the styloglossus, hypoglossus and genio-

glossus muscles, in close proximity to Wharton's duct of the submaxillary gland.

The mucosa of the alveolar ridge of the mandible is normally thinner at the crest of the ridge than it is on the labial or lingual slopes. Therefore, the crest of the alveolar ridge should not have, as it cannot withstand, the amount of pressure that may be applied to the sloping sides. Just the opposite is true in the maxilla, where the alveolar ridge is so covered that it is capable of withstanding the force of mastication. The mucosa overlying the lingual nerve, as it lies medially from the mylohyoid line in the region of the third molar, is usually very thin, and, in most cases, the pressure exerted by the denture must be less than the pressure applied elsewhere. The mucosa and underlying structures in the lateral pressure area are capable of withstanding a fair amount of pressure, which, being lateral, is unaffected by masticating force and remains constant at all times. Too much lateral pressure would result in unbearable pain, and too little would result in loss of peripheral seal. The pressure applied is best tested in the rebased denture by placing the index finger over the incisors and tipping the denture linguolabially, when the denture in the lateral pressure area should move up and down at least 3 mm.

To get an impression of the entire area desired for the lower denture, it is necessary to take the lower impression in sections, it becoming, at its best, merely a preliminary impression. A stock tray is selected, long enough distally to include the retromolar triangle, and the distolingual curvature of the tray trimmed to an angle of 45 degrees to the occlusal plane, along the area of the mylohyoid line. A compound impression is then taken of the ridge, the operator determining that the compound has included all the desired peripheral tissues. After the chilling and removal from the mouth, the distolingual portion of one side is cut back to the border of the tray along the trimmed edge. A piece of softened compound the size of an olive is tacked to this border of tray and compound; the impression is seated, and, with the index finger at right angles to the cut border of the tray, the compound is forced gently under about 5 pounds pressure into the lateral pressure area. The material is chilled and removed. A groove is cut in the compound on the back of the impression along the trimmed border of the tray, and the added extension is broken off cleanly and laid aside. The opposite side of the impression is completed in like manner, and the broken side is then replaced and attached with sticky wax. The impression is now ready for the pouring, which is done with ordinary plaster.

After separation, the cast is ready for the study relative to marking on it the anatomic outline of the denture. The tendency should be to overbuild the biteplate so that it may be trimmed back to toleration of the functional range of the border tissues. Therefore, labially, the outline is marked well into the soft tissue attachments. Buccally, the outline should be extended well toward the external oblique line. On the lingual aspect, the outline should dip as far as possible below the second bicuspid area, proceeding upward and backward along the mylohyoid line to the second artificial molar area, when, under the distal cusp area of that tooth, the outline should cross the mylohyoid line into the lateral pressure area, dipping 1 mm. and making a half circle upward to meet the buccal outline.

The biteplate is built, and, at the next appointment of the patient, the trial plate

is trimmed to toleration. It is trimmed on all borders so that the peripheral tissues in their maximum range do not unseat the trial plate. In the lateral pressure area, the trial plate is trimmed to allow the tongue to protrude one-eighth inch below the vermilion border of the lower lip, which does not allow the tongue its maximum range, but does allow all the range ever required by the patient.

The denture vulcanized to the tolerated outline must not be considered a finished denture. It is a denture with grossly inaccurate peripheral borders, with the various pressures demanded by the underlying mucosa under no control whatsoever; for example, the pressure exerted on the crest of the ridge by this preliminary denture is the same as on the sloping sides of the ridge. It is a denture with the lateral pressure arbitrarily set with a definite pressure, which may or may not be accurate for that case. Therefore, to correct the situation, the denture must be rebased; for it is not possible for anyone, no matter how skilled he may be in the handling of impression materials, to build a lower denture to the proper peripheral tissues, and, at the same time, exert the desired pressure on the tissues capable of withstanding those pressures, and incorporate the necessary relief where it may be needed. This again, it must be stated, cannot be done from a single or master impression of the lower mouth. The denture should be rebased, and rebased under a definite, equalized pressure, comparable to, or accomplished only by, perfect balanced occlusion.

The lower denture should be rebased immediately after the first vulcanization. To equalize the pressure exerted by the upper denture in centric occlusion, the teeth may either be milled to a balance, or a strip of compound laid along the bicuspids and molars of the upper denture, the compound being warmed and tempered evenly in hot water and placed in position in the mouth, and the lower then brought up into a centric relation to a point almost through the compound, thus avoiding cusp contact. The compound is chilled and the lower denture removed. Thus, it is recognized that the pressure of the upper denture in this centric relation will be equalized, and the operator may be assured the case is in the centric relation just established.

The lower denture is now trimmed, one-sixteenth inch of vulcanite being removed from the tissue surface of the denture at all points, with the exception of the lateral pressure areas. After this is accomplished, an additional one-sixteenth inch is removed from the periphery, the border being beveled parallel with the surface of the underlying tissue.

With a tracing stick, compound is now applied to the denture along the buccal, labial and lingual borders, with the exception of in the lateral pressure area; the compound is heated by means of a mouth blowpipe, or any other small flame, tempered in water and placed in the mouth, and the teeth are brought together steadily in centric occlusion. The application of the compound along the border allows the crest of the ridge to be free of pressure, as the compound compresses the tissues on the sloping sides of the ridge, flowing to meet the compound of the opposite side at the crest of the ridge, and flowing under the border of the denture as excess. The denture is removed, and any imperfection in the flow of compound is corrected by the addition of compound and reseating under pressure.

The excess of compound on the labial and buccal is trimmed away with a sharp knife, and the border warmed and re-

seated in centric occlusion. The patient is then instructed to pucker the lips, as in the act of kissing, then to pull back the corners of the mouth as in a wide grin. This brings the border tissues, including the buccinator muscle, into the patient's functional range, and trims the compound accordingly by folding or rolling it up over the buccal and labial margin of the denture. This is repeated until no excess is noticeable on the buccal and labial periphery.

The excess is then trimmed away accordingly on the lingual aspect, the compound warmed and tempered and the denture seated in the mouth. The denture is held in the mouth by finger pressure, and the patient is instructed to extrude the tongue and also to elevate the tongue to touch the roof of the mouth. This action of the tongue produces the functional range of the lingual frenum; the coordinated action of the genioglossus and the geniohyoid muscles raising the sublingual gland against the margin of the denture, and also the functional range of the mylohyoid muscle. The denture is removed and the process repeated until all excess disappears, and an accurate anatomic border crosses the mylohyoid line, onepletely rebased, with the exception of the lateral pressure areas, which are rebased singly. The only part of that area of the vulcanite denture to be trimmed previous to rebasing is the border itself. Beginning at a point on the lingual margin anterior from the place where the denture border crosses the mylohyoid line, one-sixteenth inch of vulcanite is removed along the border around the half circle over the mylopharyngeus to the retromolar triangular area, to allow the tissues to form a compound border to the denture in this area on extrusion of the tongue, and also to give relief over the area in which the lingual nerve comes in greatest proximity to the overlying mucosa.

The vulcanite in the lateral pressure area is now covered with compound and the denture seated in the mouth in centric occlusion. The mouth is opened, and the denture held in place with finger pressure, while the patient is instructed to extrude, elevate and move the tongue from side to side. The denture is removed, and the process is repeated until all excess has been removed, and the border is in harmony with the peripheral tissues. The size of this area, as has been explained previously, is dependent entirely on the action of the palatoglossus muscle and its curtain-like soft tissue attachment to the mandible. In a Class 3 case, the area would be small; in a Class 1 case, the area would be large. The perfect half circle form of the border should always be maintained.

The corresponding area of the opposite side is then prepared and rebased the same way. Then, the entire seating area with the exception of the lateral pressure areas is but slightly warmed by a gentle painting with the flame, dipped in water and placed in the mouth, and the patient is instructed to close in centric occlusion. The denture is now tested for lateral pressure by attempting to tip the denture labiolingually. If so much pressure is being exerted that the denture does not move, it should be removed from the mouth, scraped slightly where the lateral pressure is being exerted, but not at the periphery, warmed slightly and returned to the mouth. This should be repeated until the denture will respond to the tipping force by moving up and down at least 3 mm. in the lateral pressure area.

The rebasing technic is now completed and the denture may be tested for stability and seal. It will be noted that the denture is in complete harmony with the

functional range of the border tissues, that the desired pressure has been applied and controlled in the areas capable of withstanding pressure, and those areas demanding relief have been treated in accordance with their anatomic and histologic structure.

In review, may I outline the peripheral border of the upper denture for those who wish to build dentures to soft tissue as follows: If we start at the crest of the ridge in the central area, passing labially, the first soft tissue encountered is the frenum of the lip. In the lateral area, we go beyond the frenum to the fold or curvature of the soft tissue to the lip. The same holds true for the cuspid area. Above the two bicuspids, one usually finds accessory attachments of the buccinator muscle, along with strong plicae of soft tissue. The first molar area indicates the attachment of the buccinator muscle. The second molar area is bordered by the soft tissue fold of the cheek. There being obviously no lingual border of the upper denture, the peripheral border is formed by the posterior plate line, which is determined by definite anatomic landmarks. This area, as noted before, is divided into five sections from tuberosity to tuberosity, Areas 1 and 5 from the buccal aspect of the tuberosity medially through the triangular notch between the tuberosity and the hamular process; Areas 2 and 4 marking the insertion of the palatinus. Area 3 is the midline area distally from the border of the palate bone, and anteriorly from the border of the movable tissue of the soft palate.

The periphery of the lower denture is somewhat more complicated. If we start, as before, at the crest of the ridge in the central area and passing labially, the first soft tissue met is the frenum of the lip. The lateral and cuspid areas are similar in that they pass labially from the soft tissue of the lip. The bicuspid area is marked as in the upper denture by strong plicae of soft tissue and by auxiliary attachments of the buccinator muscle. The border in the molar area goes buccally toward the external oblique line to the broad attachments of the buccinator muscle. On the lingual aspect, the central area passes downward to the frenum of the tongue. The lateral area is bordered by the anterior portion of the sublingual gland. The cuspid area is bordered by the middle or highest portion of the sublingual gland as it passes upward and backward in a gentle curve, passing downward and backward beneath the first bicuspid area. The deepest point in the lingual outline of the lower denture is reached at a point directly underneath the second bicuspid area, as that point marks the small space between the distal end of the sublingual gland and the first noticeable portion of the mylohyoid line and its muscle. The first molar area is bordered by the functional range of the mylohyoid muscle as it passes upward and backward underneath the second molar area to the distal cusp of that tooth, where the border of the denture slips over the mylohyoid line, as there are no muscle attachments on the mylohyoid line from the distal end of the mylohyoid muscle to the mesial end of the mylopharyngeus.

The outline of the denture then dips 1 mm. and continues downward, upward, and forward slightly in a perfect half circle, the size of which is dependent on the action of the soft tissue curve from the mandible to the palatoglossus muscle, which is the anterior pillar of the tonsil. The outline continues over the mylopharyngeus and the pterygomandibular raphe attachment on the medial border of the retromolar triangular area and passes well distally over the retromolar triangular pad of soft tissue overlying the ter-

minal fibers of the temporal muscle, joining the buccal border at this point.

Thus is the field in which we work composed, not as the result of opinion or hypothetical surmise but of actual dissection. This is the anatomic structure with which we, as denture men, are working every day, and it necessitates only our careful endeavor to work in harmony with it. While the anatomic structures, as outlined, appear with almost unvarying constancy, it becomes apparent that one must thoroughly know the normal in order that one may recognize both major and minor abnormalities. The ability of any operator to recognize the abnormal in anatomy varies directly with his knowledge of the normal; that is to say, the less his knowledge, the greater his ability to recognize the abnormal only by its grotesqueness, minor abnormalities, which may interfere greatly with the success of the case, slipping by unnoticed and unrecognized. Therefore, what I hope to have accomplished is to review the anatomy as we were taught it in our undergraduate days, to bring back to mind the relation of the various anatomic structures involved in the seating areas, and to emphasize the fact that there is a definite anatomic outline, regardless of the technic used, for those who wish to build the periphery of the dentures in soft tissue. Building to this outline, applying pressure and relief in harmony with the resiliency of the underlying tissues, with an equal effort to secure and establish a balanced occlusion, and to produce pleasing effects, not only enables the operator to discharge faithfully his professional responsibility as regards the patient's comfort, health and happiness, but further affords that added satisfaction that comes with the knowledge of a piece of work well done.

DISCUSSION

Clyde H. Schuyler, New York City: Too few of us have an appreciation of the value of examination of denture borders in the mouth, complete examination and charts of the case, before starting the construction of dentures. I think there is no way that one can familiarize himself so thoroughly with mouth conditions as by having a complete chart for each individual mouth. In many instances, we perhaps enter into the construction of a prosthetic appliance and then find difficulties that should have been observed before we started. A thorough examination of the mouth is impressive to a patient and aids us in acquiring his confidence. There are many things essential to success in full denture construction. One may be a master of the handling of plaster compound or wax, but if he does not have a thorough knowledge of denture areas and the tissues he is dealing with, he cannot expect the ultimate of success.

Ewell Neil, Nashville, Tenn.: The upper technic has not been changed in about twenty years, except for a change made about twelve years ago. The lower technic had its inception in the Meharry Dental College of Nashville, Tenn. The clinical material offered in the anatomic laboratory of that great school made it possible for us to start this technic.

ROUND WIRE CLASP AS APPLIED TO PARTIAL DENTURES*

By LEO G. POLLOCK, D.D.S., New York City

IN February, 1924, Henry W. Gillett[1] introduced a form of partial denture restoration which employed the round wire clasp for retention and a stabilizing lug to counteract the forces of mastication. I should like you to consider my paper as a supplement to his, and I shall also endeavor to give you the technic for the construction of such a partial denture. In Dr. Gillett's paper, the advantages of this restoration over other types of removable appliances are stated so clearly that a brief review will provide a background for a clear understanding of this subject. I am in full accord with Dr. Gillett's opinions, except where such differences are stated.

Dr. Gillett dismisses the fixed bridge as being inadequate except in small cases because of the "inability to keep it clean enough to insure stability of abutments and health of gingival tissues, within the limits of time and effort that patients are willing to give to it."

The buccolingual precision attachments, in order to have sufficient strength to endure for some time, must necessarily be large, and therefore require cavities so deep at the cervical portion that the pulp is endangered, especially when cavities are prepared in previously sound teeth. He also calls our attention to thermal shock from large inlays which have a "driving fit" to hold these inlays against the pull of the attachment.

The presence of extension attachments which overlie the gingival tissue, in addition to the terrific strain on the abutments, makes the maintenance of cleanliness more difficult.

The wide cast clasp, while an adequate retaining device, causes so much destruction of enamel in most mouths that its use should be emphatically decried.

In summing up, Dr. Gillett says:

> It is self-evident that whatever risks there may be of malinfluence on pulps of vital teeth, by reason of the size of inlays needed, or their close approach to pulp and sensitive areas in the case of their use as receptacles for the box elements of split attachments, this feature will disappear, if split attachments for vital teeth be discarded and the retainers used be applicable to the exterior of the tooth. It is also self-evident that from the standpoint of exercising an influence favorable to caries, the narrower the area of contact between any externally acting device, and the surface of the normal tooth, the less the risk. This, of course, points at once to the clasp made of round wire, of an alloy capable of maintaining an elastic tension, for long periods, without noticeable deterioration.

It must be borne in mind that round wire clasps of small gage having great elasticity will fall short of the mark unless we use with them stabilizing lugs of such design that stresses due to the forces of mastication as well as lateral strains are counteracted. This at once throws in

*Read before the Section on Partial Denture Prosthesis at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 22, 1930.

1. Gillett, H. W.: Dent. Items Int., 46:126 (Feb.) 1924.

the discard the occlusal rest placed over the marginal ridge of the abutment teeth. There are selected places where an occlusal rest may be used in conjunction with the stabilizing lug, but they should never be used alone.

The rectangular lug relieves the clasp of much of the demand made on it if no lug is used; or if the occlusal rest is selected, the lug takes care of the stresses so that the only function of the clasp is to support upper pieces against gravity and all of them against traction of adhesive foods. But little retentive force is required for this.

The stabilizing lug should have a definite rectangular form with well-defined straight sides and floor but somewhat smaller at the cervical than at the occlusal surface. The lug must have a seat in an abutment restoration, which usually should be an inlay.

At once, the advocates of the precision attachments raise the point as to why, if we must have inlays, we should not insert a precision attachment and do away with the clasp entirely. I admit that when the abutment teeth have large deep cavities or large fillings or inlays of long standing, so that little if any new cutting is necessary, and if the crowns of the abutment teeth are not too short, the precision attachment may be used to advantage.

In the case of sound abutment teeth, there is a great difference. An inlay cavity can be prepared which is so shallow at the cervical portion that the smallest (and therefore the weakest) precision attachment cannot be inserted within its border, and yet there will be ample room in which to place a lug seat in an inlay made for the same cavity. It is this conservation of vital tooth substance at the tooth's most vulnerable point which dictates the selection of the lug and round wire clasp as the ultimate attachment in all cases where the abutment teeth are sound.

Dr. Gillett advocated the use of 16, 17, 18 and 19 gage round wire. Since that time, research in gold alloys containing high percentages of platinum and metals of the platinum group has made it possible to use smaller gage wire, chiefly 19, 20 and 21.

It is fitting that we pause for a moment and pay tribute to the memory of the late Louis J. Weinstein, whose researches in dental metallurgy lately conducted in association with the Bureau of Standards at Washington, D. C., have made these improvements possible. His contributions to prosthetic dentistry have been numerous and his untimely death leaves a place in dental research which it will be difficult to fill.

What is the advantage in using smaller gage wire?

One of the governing laws of elasticity is that deflection varies inversely as the fourth power of diameter. Another is that deflection varies as the third power of length. Translating this into practice, we find that the smaller the wire and the longer the clasp we can use for an individual case, the greater the deflection possible, and consequently the greater the factor of safety for a small deflection. This, in turn, calls for a wire of the highest possible elasticity and the greatest adaptability to heat treatment, because a small wire with these qualities will have the same retention value as the larger wire without them. And, in addition, it can be depended upon to display the required amount of deflection to permit the clasp to pass over the contour of the tooth, and close up again to the desired smaller size, a great number of times without permanent deformation and consequent loss of retention value.[2]

TECHNIC FOR DENTURE CONSTRUCTION

Let us now take up the construction of a typical two-armed clasp denture where a first molar and second bicuspid

2. Gillett, H. W.: Retainers in Partial Prostheses, J. Dent. Res. (Sept.) 1927, p. 189.

are missing and the adjacent teeth are sound. The preliminary survey having been made, and this type of restoration selected, typical inlay cavities are prepared in the distal portion of the first bicuspid and the mesial of the second molar. By typical inlay cavities, I mean cavities which are not any deeper or wider than would be employed if these teeth had the smallest interproximal cavities that could be detected by a roentgenogram. Indirect cavity impressions and a wax bite are taken and the cavities closed with a temporary filling. When the wax patterns are being constructed in the laboratory, small rectangular seats are carved out of the wax and the inlays are cast, finished and polished. Inlay gold should be the very hardest that can be obtained. Metallurgists have proved that no gold has yet been alloyed which is so hard as dental enamel; and recently some of the leading periodontists have stated that there is less traumatic occlusion from hard gold than from the soft golds which flow under the stress of mastication, and do not wear down so readily nor so uniformly as the harder alloys.

The floor of the lug seat should be inclined slightly rootwise at its axial end, and the side walls should also be inclined somewhat to give a tapering form, this making the lug smaller at the cervical than at the occlusal part. The general form of the lug seat is such that the lug enters its seat easily and yet tends to remain seated when in place.

The lug patterns are then waxed on the finished inlay, leaving an apron about 1 mm. wide on the proximal surface. The sprue is inserted in the occlusal surface, and the lug is cast. In cutting the lug from the residue button, a short portion of the sprue is left attached to the lug. This will serve as a handle and also as an aid in seating the lug in a plaster impression.

The patient is now seen for the second appointment. The inlays are adjusted for occlusion and cemented into the cavities, and another wax bite is taken. The lugs are then waxed in their respective seats. I dry the lug seat in the inlay; and grasping the handle of the lug with a pair of pliers, gently heat the lug in a small bunsen flame, and touch the cavity side of the lug to a stick of wax, allowing only a small quantity of wax to adhere to the lug, and quickly place in position in the inlay. For this, I use a sticky wax that is not too adhesive and will allow the lug to come away with the plaster impression, being certain to get an accurate impression of the teeth to be clasped. At this time, I also take the shade.

The lugs are now replaced in the impression, if they have become loose, and all traces of wax removed from them. Fusible metal is poured into the abutment teeth, care being exercised so that no metal runs onto the saddle area. Small iron loops should be inserted into the fusible metal to secure it in the model. After the metal has been cast, the remaining surface of the impression is coated with shellac and then sandarac and allowed to dry thoroughly. The impression is then poured, a good artificial stone being employed.

You are probably wondering why the impression was not taken with the inlays uncemented so that the bridge could be constructed directly upon the inlays in the model. There is a twofold reason for this: (1) the cementing of the inlay after the case is constructed would tend to raise the denture, perhaps only slightly, but this is entirely eliminated by setting the inlays before taking the impression; and (2) the slight film of wax on the lug allows for just that amount of compres-

sion when the denture has been completed.

The impression is then separated, the lugs are removed from the model and the case is articulated. The porcelain crowns are ground nearly to fit the model, where the saddle will later be constructed, and approximately to occlusion, being left a little longer than required to allow for the final adjustments. A space equal to the gage of the wire to be used for the clasps must be left between the porcelains and the abutment teeth. This space will be occupied by the uprights. A plaster key is made to hold the porcelains in place on the model. The porcelains are now ground for the boxings, a space about 26 gage being left between them and the model for the saddle.

The saddle section, consisting of the saddle, the boxings for the porcelain crowns and the uprights is now formed in wax on the model, the plaster key being used to hold the porcelains in place in so doing. This section is cast with a hard elastic gold and rough finished but not polished.

The saddle section is then placed back on the model and the lugs are now to be soldered to the section. It is advisable to solder one lug at a time. The small handle on the lug is again useful in steadying the lug in the soldering investment. After the lugs have been soldered and all joints between lugs and section nicely finished and polished, the lug handles are ground away and the denture is ground to exact occlusion.

With a suitable bur, a groove is cut across the occlusal aspect to the uprights. This groove is to be just wide enough and deep enough to house the round wire clasp.

We now come to the bending and shaping of the clasps, which is not a difficult task but one which must be carried out with careful attention to details.

The first step is to secure round wire which has been carefully manufactured and accurately drawn. A suitable length of the wire (gage 19 for the molar and 20 for the bicuspid) is quenched. This renders the wire soft and pliable so that it can be bent to shape without being nicked or unduly compressed at any point. It is most important to use pliers from which all sharp corners have been removed by grinding.

I like to begin the bending of the clasp by forming the lingual arm, which is placed near the gingival margin but not impinging it. This is tried against the fusible metal tooth and the wire bent to form the vertical segment of the lingual arm. Each time a bend is made, the wire is brought back to the model and held securely in place, to determine that each segment fits properly. The vertical segments of the clasp must not touch the model. They must be kept in the center of the embrasure and slightly away from the metal tooth. The next bend is a right angle to form the occlusal segment of the clasp. This segment fits into the buccolingual groove which was made on the occlusal aspect of the upright next to the lug. Now we bend the wire to form the vertical segment of the buccal arm, and finally bend it to fit into the angle formed by the buccal surface of the tooth and the gum margin, being careful to keep it from impinging on the gum. Especial care is needed to avoid allowing the angle between the horizontal and perpendicular segments to bear against the gum.

You will observe that the clasp has an occlusal segment, a buccal and a lingual vertical segment extending from the occlusal to the gingival border of the tooth and a buccal and a lingual horizontal segment at nearly right angles to the ver-

tical segment. When the clasp is opened to pass over the highest convexity of the tooth, the horizontal arms exhibit flexure elasticity. The vertical arms are also flexed to a slight degree, but bring into play another form of elasticity known as torsional elasticity, which is even more important than flexure elasticity.

The completed clasps are then placed in their respective grooves and a small plaster key is made which covers the clasps and saddle section. Before the clasps are set back into the key, they should be boiled in 30 per cent nitric acid to remove all traces of the fusible metal with which they would otherwise become contaminated. The key containing the clasps and saddle section is treated like an impression, and a soldering investment model is made from it. The clasps are soldered at the point where they rest in their grooves. A very small piece of solder is used and just enough heat to flow it. We must not overheat the case nor unite any more than the occlusal segment of the clasp. If, perchance, any solder flows onto the vertical segments, the clasp has been spoiled and will have to be detached and discarded.

After the clasps are soldered to the saddle section, minor adjustments in the fit of the clasps are made and the denture is again boiled in 30 per cent nitric acid. This brings us to the final operation of producing maximum resiliency of the wire, or "heat treatment."

HEAT TREATMENT

Until recently, this maximum resiliency was obtained by heat treating the finished denture in an electric oven. During the past year, I have been allowing the case to cool slowly in its investment after the final soldering.

Several years ago, through the courtesy of Mr. Coleman, then of the Bureau of Standards, I was permitted to make many tests with wires in the quenched, air-cooled and oven-cooled states. Some of the materials tested showed remarkable improvement after heat treatment. This same wire when soldered to the denture and heat treated in my laboratory became extremely brittle. Undoubtedly, heat treatment has a marked effect on these elastic wires, and further research is needed to produce a definite technic which will give maximum resiliency without brittleness. John S. Shell[3] says, "The problem of heat treatment of dental alloys will be found eventually to be a rather complicated procedure, requiring a special technic for each alloy."

The oxids which have been formed on the denture while it was being soldered may be removed after heat treatment or air-cooling by boiling in dilute hydrochloric or sulphuric acid. This does not affect the properties of the wire.

In a one or even a two tooth denture such as I have described, one can dispense with the buccal arm of the bicuspid clasp if it should be too obtrusive.

In designing clasps, we should remember that only the final third of the horizontal segment should make contact with the tooth. The vertical segments must never come in contact with the tooth. If a vertical segment of a clasp impinges on the tooth in either removing or inserting a denture, the clasp will be unduly strained and its life will be short.

An important clasp is the "Gillett crib," which is attached to the saddle at its lingual end and rests in an occlusal embrasure and has its buccal arm free. This type is generally used with a cantilever saddle where only one definite lug seat can be obtained.

3. Shell, J. S.: Heat Treatment of Gold-Nickel Alloys, J. A. D. A., 17:409 (March) 1930.

The variety of shapes into which the round wire clasp can be easily bent to overcome tipped abutments and extreme convexities of crowns make it an excellent attachment to be used in conjunction with many other forms of retainers such as the split pin or bar and the buccolingual precision devices.

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DISCUSSION

F. M. Hight, Houston, Texas: I agree with Dr. Pollock in most of his statements as to the use and advantages of the wire clasp. I am sure that we have all watched partial dentures with wire clasps long enough to know that they function pretty well. There is not anything perfect in dentistry, and of course improperly made partial dentures with wire clasps are going to give trouble. There are men today that are making one-piece castings that live up to practically every fundamental that Dr. Pollock has shown. One-piece castings can, I think, be cast in such way that the frictional contact or contact with the clasp against the tooth will be no greater than with the wire clasp, as has been shown. One thing on which I would differ with Dr. Pollock is the use of rectangular grooves in the teeth. He uses the flat seating base, which necessitates the placing of a gold inlay in each tooth that he uses the rest on. The reason that I would not agree to this placing of rectangular cavities in there is the fact that I believe we might just as well place a stationary bridge in these small cases, because, as far as the strain is concerned, we will get all the strain on the abutment teeth with this type of lug that we would get with a stationary bridge. In my own practice, I would prefer a lug which fits into a seat, preferably ground into the enamel, provided the enamel is thick enough, where the occlusion will permit the placing of a lug, and I think that a lug placed in a saucer shaped groove, placed in the tooth so that the partial denture or the added teeth may move and the lug may move also, causes less trauma to the abutment teeth. According to the pictures shown here, Dr. Pollock is using wonderful wire. I have had much trouble with wire clasps breaking when the expansion or bending occurs.

Dr. Pollock (closing): There is not anything wonderful about the wire that I use. Any good wire will, if properly used and not abused, permit one to make all the necessary bends in them that are required. Of course, we must employ a wire which, when quenched, will be quite soft. If the wire is soft enough, we can bend it. With the exception of the little loops, most of the loops are bent by hand, just bent around an orange wood stick and forced on the metal. The wire is very soft when it is annealed or quenched, and after the final soldering for heat treatment, it retains its original elasticity. There is no trick to that. It is very simple, and any good wire will do that. I have been working to reduce the size of the wire that I use. As stated in the text, at first I used 16 and 17 gage wire. I am now using 19 and 20 gage for molars and bicuspids, 18 gage for crib clasps and 21 gage for closed crib clasps.

BACTERIOLOGY OF VINCENT'S INFECTION

By FRANCIS H. DALEY, D.M.D., Boston, Mass.

IN a study of the bacteriology of any disease, there are certain fundamental principles which must be followed. The cultural and morphologic characteristics of the organisms must be studied. Their relation to certain lesions must be definitely established. Whether they tend to be saprophytic or parasitic is a factor. Some organisms liberate exotoxins. The Klebs-Loeffler bacillus is an example of this type. Other organisms store up their toxic materials as endotoxins. An ex-

ample under this classification is the typhoid bacillus.

Heretofore, one of the most important factors in proving that certain organisms produce certain effects or lesions was the fact that they follow the fundamental principles known as Koch's postulates. This theory, while fundamental, has been subject to modification in recent years. Koch brought out the fact that to determine whether an organism was the causative factor in a particular pathologic process, it was necessary that these postulates be satisfied in sequence. The organisms must be found constantly in the lesions. They must be taken from the lesions and grown in pure culture. The organisms from the pure culture must be inoculated into susceptible animals and produce lesions identical with the first. The organisms from these experimental lesions must then be recovered in pure culture. Most organisms associated with pathogenic processes will follow these principles. There are some few which do not adhere to them in every detail. The spirochete of syphilis is one of the more important organisms in this classification. Until very recently, it has been practically impossible to produce the lesions of syphilis in experimental animals by strict application of these laws. It is now possible, with some modification, to prove that the organisms are directly responsible for the lesions of the disease. The organisms were found constantly in the lesions, so that there was no question as to their being the causative factor. So it has been with Vincent's infection. The organisms have been constantly in the lesions of the disease; so that, until it is definitely proved that they are not the causative factor in its production, we must accept them as being allied with the disease.

In discussing Koch's postulates, we must not overlook the fact that there are some diseases which necessitate the use of the human being for the production of the secondary, experimental lesions. All attempts to successfully inoculate lower animals may fail, but infection of another human being will result in the production of typical lesions from which the organisms may be recovered. Such was the case in experimental work with the gonococcus. The relation of the organism to the disease was not definitely proved until experimental work was done on insane women. Therefore, when dealing with Vincent's infection in experimental animals, we may be dealing with nonsusceptible animals. It seems certain, from clinical evidence, that an individual suffering with an acute Vincent's infection may inoculate another susceptible individual, with the result that similar lesions and organisms appear in the second.

In considering the causative agents in Vincent's infection, we are concerned with two organisms. Where two organisms are involved in the production of a disease, the relation of one to the other must be carefully considered. Symbiotic action is always a factor in such cases.

Symbiosis is a state in which two organisms live, grow and reproduce to the mutual advantage of each other. The life processes of one type may or may not be independent of those of the other. Dependent or independent, the life processes of the one do not interfere, in any way, with the fundamental functions of the other. When such a condition of symbiosis exists, we note the production of lesions characteristic of the disease. When only one type of organism is present, the lesions, if present, are not typical for the condition. Symbiosis may take place where complex tissue or food products are split by one species to furnish nutritive material for ingestion of the

other species of a lesser digestive ability.

Varney has shown definitely that the fusiform bacillus in pure culture is odorless. This would lead us to believe that symbiotic action is not a remote possibility. The odor associated with Vincent's infection is characteristic for the disease, and is no doubt due to the splitting up of tissue and food materials by bacterial action. Bacteriologically, it is well understood that certain chemical effects, such as the decomposition of organic material, cannot be produced, in many instances, by one form of bacteria alone—such action occurs only where two or more species may be associated.

Consideration of the species of organism that we are dealing with is a factor. The terminology, particularly in regard to the spiral forms, has been more or less obscure. Bergey, in his latest classification, lists the spiral organisms of Vincent's infection as *Borrelia vincenti.* Bergey says: "Spirochaeta, borrelia and treponema are distinct genera of the family, Spirochaetacae. The genus spirochaeta, however, is usually saprophytic, free living in water and with the protoplasm spirally wound around an elastic axis filament. Referring to Spirochaetacae which are normally found in the oral cavity as spirochaeta, is, therefore, incorrect." We shall, therefore, in discussing these organisms refer to them as *Borrelia vincenti* and *Fusiformis dentium.*

It has been shown that many of the bacteria which are capable of producing disease in man can and do live as saprophytes. That is, they may live apart from a living host and can obtain all the nutrient material necessary to growth from dead organic material or from simple organic salts and water. The distinction between "biogenic" and "pathogenic" bacteria is a relative one, dependent, in many cases, on environmental factors coupled with tissue-cell changes from some local or remote etiologic source.

Veillon and Zuber were probably the first investigators to do any organized research work on the bacteriology of the organisms. This was in 1898. From that time on, much was done, and the more prominent names associated with this work were Abel, Lewcowicz, Ellermann, Tunnicliff, Weaver, Krumwiede and Pratt, Valentine, Brams, Pilot, and Davis, McConnell, McNiell, Knoor, Utz, Muhlens and Hartmann, Kritschevsky and Seguin, and Varney.

The work of Philip L. Varney, of St. Louis, in 1927, is probably the most authoritative of the later publications. His work contains a complete history of the investigations of other men and his review and the conclusions drawn from his own work seem rational and free from bias.

Varney, by means of serum agglutination tests, has isolated three main groups and two subgroups of the fusiform bacillus. By morphologic differentiation, he has added a fourth group to the series. He has classified them as follows: (1) filamentous; (2) slender; (3) broad; (4) wavy.

He found that the best culture medium for the growth of the fusiform bacillus was blood-agar. The organism was incubated on this medium, in anaerobic jars containing phosphorus, for a period of forty-eight hours. He believes that the high moisture content resulting from the use of phosphorus greatly aids the growth of the organisms. Varney shows that when a culture gives off a foul odor, impurity is indicated. He has also shown that even slight changes in the composition of the medium will result in marked morphologic changes in the organisms.

Varney, in experimenting on the growth of the fusiform bacillus, from surface cultures, found that if the blood-agar medium contained a small amount of arsenic, the borrelia would be eliminated. By careful adjustment of the arsenic content, he was able to kill the associated organisms and keep the fusiform bacilli alive. He was then able to transplant them and grow them again, provided no more arsenic was used. These findings were misinterpreted by the Drs. Greenberg of New York in an article published in the *Dental Cosmos* in April, 1929. They said: "He [Varney] has ruled out the symbiotic nature of the spirochete and fusiform bacillus by growing both germs on an arsenic medium. Only the fusiform bacillus grew out in each case." Varney, in one of the first paragraphs of his paper, says: "Fusiform bacilli grow in symbiosis with other organisms to a greater extent than has been realized. Some of these contaminating organisms may be so small as to be mistaken for débris, especially when liquid cultures of the organisms are studied."

Varney has found all types of the fusiform bacillus to be nonmotile and gram-negative. He has found the filamentous type growing up to 225μ in length. The average for the filamentous type was from 4 to 19μ in length and from 0.4 to 0.7μ in width. The slender type averaged 3.9μ in length and 0.58μ in width. The wavy type averaged 5.5μ in length and 0.5μ in width. The broad type averaged 12μ in length and 0.9μ in width.

We have taken Varney's work as the standard in bacteriologic research on the fusiform bacillus because, after a study of most of the literature on this subject, we feel that he has given the clearest and most comprehensive analysis, as well as the most scientific development and presentation of the material that is to be found in the literature.

Bacteriologic research on the borrelia of Vincent's infection has been completely ineffectual as far as scientifically positive results are concerned. There is considerable controversy as to whether the organisms have ever been grown in pure culture. Muhlens and Hartmann claim that they have grown them in pure culture. They say that no pathogenic lesions followed inoculation of animals with these organisms. There is so much chance for error when dealing with spiral forms from the oral cavity that we cannot be sure of their findings until it can be definitely proved that they are the same forms which are associated with Vincent's infection.

Kritschevsky and Seguin have inoculated guinea-pigs with material from a pyorrhea pocket. This material was rich in the organisms commonly associated with Vincent's infection. They found that such inoculation resulted in abscess formation. They also noted in the lesions so produced, the borreliae were preponderant in numbers over the fusiform bacilli. It has been our observation in clinical studies of smears from human lesions that the borreliae always greatly outnumber the fusiform bacilli.

In dark field illumination of smears from Vincent's lesions, we have always found the fusiform bacillus to be a nonmotile refractile organism. The borrelia is longer than the spirochete of syphilis and averages from about 8 to 18μ in length. It is decidedly thicker than *Spirochaeta pallida,* and, in most cases, seems to be thicker than any of the other spiral forms found in the mouth cavity. It averages from about 0.5 to 0.75μ in width. The average borrelia has from four to ten flattened irregular spirals. It stains quickly and evenly. Its movements

are very rapid. There is some controversey as to whether the organism is flagellated.

We have noted, in studying cases which have been treated over a period of time, that the fusiform bacillus apparently does not take the stain so deeply or so evenly as the younger forms. We have also noted in these cases that the borreliae tend to take on a beaded appearance. This may be due to the fact that the treatment alters the cell content of the organisms, producing aberrant or involution forms.

Tunnicliff, in her work on the fusiform bacillus, firmly believed that the borrelia was an offspring of the fusiform bacillus. This theory has now been discarded, because many of the later investigators have shown that if the fusiform bacillus has been obtained in pure culture, the spiral forms will not appear even after two score generations have been planted and grown. Varney has shown that certain wavy forms will appear, but these are true fusiform bacilli and not the spiral forms which are associated with the disease.

There is no question that more experimental work is necessary on these organisms before it can be definitely determined whether they are the causative agents in the production of the disease. It seems to us that until investigation proves that the two types have no bearing on the disease, we must needs accept the hypothesis that they are related to the lesions of Vincent's infection. A study of the borrelia and the fusiform bacillus and their relation to the reticulo-endothelial system might prove of interest. A study of the relationship of the bacteriology of pyorrhea and of Vincent's infection is interesting, and it seems to us that in many cases the connection between the two types of lesions is more than a casual one.

HISTOLOGIC STUDY OF THE INFLUENCE OF FUNCTION ON THE HUMAN PERIODONTAL MEMBRANE*

By RUDOLF KRONFELD, M.D., Chicago, Ill.

THERE are two ways of determining the influence of function on the periodontal tissues. One way is to create decided functional conditions in experimental animals and then to section the teeth together with their surroundings and to compare them with control teeth. We can produce an excess of stress in either a horizontal or a vertical direction by means of putting crowns or caps on some of the teeth, or we can entirely eliminate the influence of function by grinding the teeth until they are out of function or by eliminating the antagonists. This kind of investigation is restricted to animal experiments.

In human material, we have to choose another method to determine the influence

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of function on the periodontal membrane. Among the jaws obtained by necropsy, we always find some in which the functional conditions existing during life can be easily determined. Occlusion, loss of antagonists, amount of abrasion, and size and distribution of the worn facets of the tooth allow us to draw definite conclusions as to the functional conditions that were present during life.

The first to make investigations of this type was Gottlieb,[1] who analyzed the histologic findings on human teeth of different ages that had for a long time been subjected to heavy stress in a certain direction, and showed how the force and direction of this stress could be determined

Fig. 1.—Anterior part of the jaws.

in the histologic sections from the arrangement and distribution of the cementum on the root surface. Orban[2] lately described a case of a man, aged 25, in whose jaws the tissue changes caused by the tipping of some of the teeth which were thus exposed to lateral stress were followed histologically. In some of these teeth, the periodontal tissues were damaged and crushed by the force of the occlusion, and resorption could be found on the tooth surface as well as on the alveolar bone. Kellner[3] studied a human jaw in which, on the one side, the upper molars had antagonists, and, on the other, had evidently been without antagonists for a long time. His findings concerning the structure of the spongy bone and the width of the periodontal membrance are of outstanding interest and importance. Kellner found that in functioning teeth, the retroalveolar spongy bone becomes reinforced; in functionless teeth, it becomes atrophic. There is a striking difference in the structure of the periodontal membrane of functioning and functionless teeth, and the periodontal space in functioning teeth is wider than in functionless teeth.

All these publications have shown us the method by which to increase our knowledge as to what changes occur in the tissues of the human jaws under the influence of function as well as of inactivity. In this paper, I wish to make a contribution to this problem and especially to show how human necropsy material can be used and analyzed for this purpose.

The jaw to be described here was obtained from a man, aged 38, who died of gastric ulcers and peritonitis. No further general nor any dental history of the case is known. The jaws were removed soon after death and fixed in formalin (solution of formaldehyd). The posterior part of the mandible is edentulous on both sides. All lower molars must have been lost long ago as the lower ridge is very flat and atrophic. Some of the front teeth, bicuspids and upper molars are still in place and have

1. Gottlieb, Bernhard: Die diffuse Atrophie des Alveolarknochens, Ztschr. f. Stomatol., 1923, No. 4. Tissue Changes in Pyorrhea, J. A. D. A., 14:2178 (Dec.) 1927 (Figs. 15 and 16).

2. Orban, Balint: Tissue Changes in Traumatic Occlusion, J. A. D. A., 15:2090 (Nov.) 1928.

3. Kellner, E.: Histologische Befunde an antagonistenlosen Zaehnen, Ztschr. f. Stomatol., 1928, No. 2.

well-formed, large crowns. In the upper jaw, the crowns of the right central and lateral incisors are destroyed by caries and only gangrenous roots are left, ending at about the level of the gums (Fig. 1). The right cuspid is a large solid tooth showing slight incisal abrasion. This tooth was hit by the antagonistic lower cuspid and first bicuspid in the lingual third of its crown (Fig. 2, above). The right upper first bicuspid is missing; the right upper second bicuspid is in almost end-to-end occlusion with the lower second bicuspid.

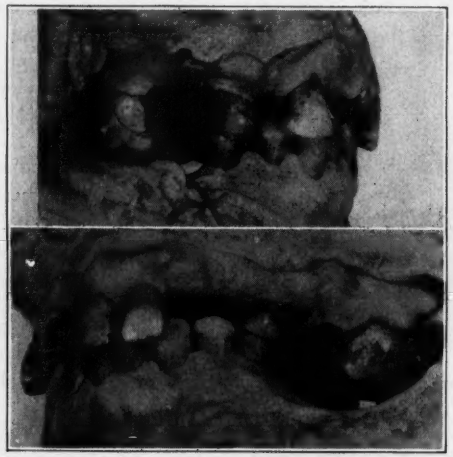

Fig. 2.—Above: Right side of the jaws. The occlusion is supported by the second bicuspids only. The cuspid is occluding labially from its antagonists (compare Fig. 1). The upper second molar is elongated and covered with calculus. Below: Left side of the jaws. The occlusion is supported by the bicuspids. The upper second molar is extruding toward the occlusal plane. The lower jaw is very atrophic in its posterior part.

The first upper molar has been lost. The right upper second molar is a large intact tooth. Its crown shows little abrasion and plenty of calculus, and it has moved down considerably toward the occlusal line. This condition indicates a lack of antagonists over a long period of time.

On the left side of the upper jaw (Fig. 2, below), there are a central and lateral incisor present. Both are intact and occlude in the gingival third of their lingual side with the lower front teeth. The left upper cuspid and first biscuspid are lost. The second bicuspid is a strong short tooth, the occlusal surface of which is worn almost flat. This tooth is in end-to-end occlusion with the left lower second bicuspid. It should be emphasized that this tooth was almost entirely free from calculus or deposits. It shows merely a slight brown pigmentation, probably due to smoking. The left upper first molar has been entirely destroyed by caries, only two short gangrenous roots being left. The second molar corresponds exactly to the right upper second molar: it is a large tooth showing no abrasion; the crown is covered with plenty of soft light chalky calculus, and the tooth extrudes toward the occlusal line as a result of inactivity of long standing. On this

side, there was also an upper third molar, which, too, shows the above mentioned symptoms of having been entirely functionless.

In summarizing the "clinical" findings in these jaws, we can say: In this case, the lower molars were lost a long time ago, and, therefore, there was no occlusion in the posterior part of the mouth. Because of this condition, the bite evidently became lowered and the upper front teeth protruded. The lower incisors, at the time of death, were articulating with the gingival portion of the upper ones. Practically the entire occlusion was supported by four teeth; namely, a pair of second bicuspids on each side which, hitting each other in end-to-end occlusion, prevented

Fig. 3.—Right upper cuspid (*C*) and second bicuspid (*B*); showing the regular dense bone structure around the latter.

a further lowering of the bite. Because of this arrangement, we can consider these four bicuspids as teeth that had to support a greatly increased occlusal stress at the time of the patient's death.

Evidently, no dental care had been exerted in this mouth at any time. The teeth decayed from caries and then were extracted, or broke down and remained as gangrenous roots. Calculus was not removed but deposited in a reverse ratio to the amount of the automatic cleaning of the teeth by chewing. Thus, all four second bicuspids are almost free from deposits, whereas, the upper molars show most of it. A roentgenographic examination of the jaws did not reveal any significant facts. As would be expected, the gangrenous roots showed large areas of bone resorption around the apex. A roentgenogram of the right upper second bicuspid, presenting a very dense, regular bone structure and a well designed inner alveolar lamina around the tooth, is shown in Figure 3.

The entire jaw was sectioned labiolingually, after being decalcified and embedded in celloidin. Serial sections were made from the material.

The teeth in this case, according to their arrangement in relation to their antagonists, can be divided into three groups (Figs. 1 and 2):

1. Teeth supporting a great amount of occlusal stress. These can be subdivided into:

(*a*) Teeth in strong end-to-end occlusion (almost axial stress): all four second bicuspids.

(*b*) Teeth exposed to a lateral (horizontal) stress: left upper central and lateral incisor, right upper cuspid, left lower central incisor.

2. Teeth in light function (in partial occlusion or in occlusion merely at certain positions of the jaws): left lower cuspid and first bicuspid, right lower first bicuspid, right lower central incisor.

3. Teeth entirely devoid of function (no occlusion in any position of the jaws): left upper second and third molar, right upper second molar.

The conditions found on the left side shall be studied first, and the teeth described in relation to their function. On the left side (Fig. 2, below), in the upper jaw we find the second and third molars entirely devoid of function. The upper and lower second bicuspids are in strong end-to-end occlusion. The first lower bicuspid has no antagonist. In some positions of

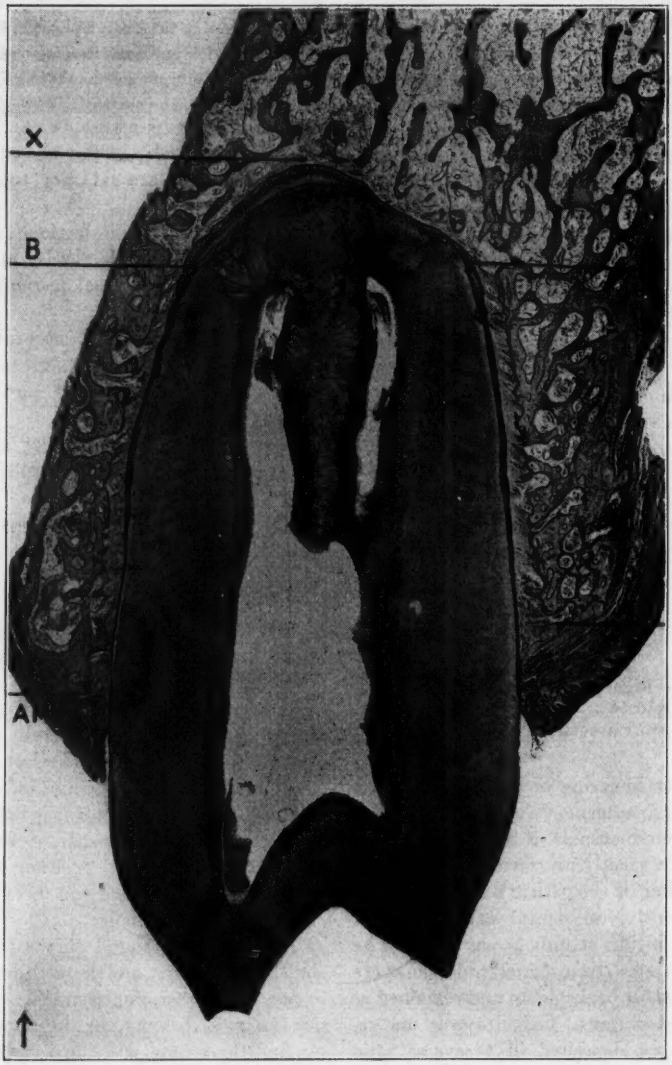

Fig. 4.—Buccolingual section through the left upper second bicuspid. (The pulp has been lost in sectioning.) *AM*, alveolar margin; *B*, border line between side and upper end of the root. The arrow indicates the direction of the occlusal force. (The lingual side is toward the right.)

the jaws during mastication, this tooth came in occlusion with the upper second bicuspid.

of group 1 (teeth supporting a great amount of occlusal stress). Figure 4 shows a labiolingual section through this

Fig. 5.—High magnification of the periodontal membrane of the tooth shown in Figure 4. *D*, dentin; *C*, cementum; *B*, alveolar bone; *F, F*, fiber bundles; *S*, spaces between the bundles containing small vessels and nerves. The arrow indicates the direction of the occlusal force.

The left upper second bicuspid will be used in this description as a representative

tooth. On the buccal cusp, the abrasion had already worn off all the enamel, and

the tip of the dentin appears slightly flattened. On the lingual side, the dentin was still covered by enamel. Most of the pulp has been lost in sectioning. The condition of the remaining pulp tissue indicates that it was healthy and normal. It can be seen, even in this low power photograph, that the periodontal fibers are very dense on both the buccal and the lingual sides of the root. They extend from the alveolar margin (*AM*) toward the nerves and blood vessels of the periodontal membrane are left between the fibers. The insertion of the bundles into the bone is dark blue in the specimen and therefore appears black in the photograph. The arrow in the lower right corner of the picture indicates the direction in which the tooth was moved when it was hit by its antagonists in end-to-end occlusion.

At the apex of this tooth, we find an entirely different condition. Figure 6

Fig. 6.—Higher magnification of area *X* at the tip of the root in Figure 4. *F*, loose periodontal tissue (irregular course of fibers); *R*, resorption of the alveolar bone; *CO*, compressed connective tissue, narrow periodontal space. The arrow indicates the direction of the occlusal force.

the apex up to the point marked *B*. This point (*B*) represents the border between the almost cylindrical root surface and the flattened root end. A higher magnification of a part of the periodontal space on the side of the root shows an unusual, dense arrangement of the fiber bundles (Fig. 5). Only very small spaces for shows the area *X* at the tip of the root from Figure 4 in higher magnification. On the left side of the picture, the periodontal space is very large. It is filled with loose connective tissue, and the few fibers that can be seen run in different directions. The bone shows evidence of resorption. On the right side of this

photograph, the periodontal space is much narrower. The fibers are arranged parallel to the tooth. The cell nuclei are not stained in this area, which indicates a partial necrosis of the soft tissue. The direction of the force parallel to the long axis of this tooth is indicated by the arrow on the lower left corner of the picture.

It will be interesting to correlate these histologic findings with the presence of an extraordinary occlusal force. The upper second bicuspid was pushed straight into its alveolus every time the patient closed his mouth. Since none of the other teeth were in sufficient occlusion to take any of the stress of mastication, the force put on the second bicuspids must have been unusually great. As a result of this stress, the fiber bundles of the periodontal membrane hypertrophied around the entire root surface. This hypertrophy can be considered as the typical reaction of a healthy, well-reacting organism to a great functional stimulus of long standing, and is comparable to the well-known hypertrophy of muscles and tendons following work and exercise (training). The dull root end was pushed upward, pressed toward the bone, and, as a result, we find relaxation of the fibers, bone resorption and compression of the soft tissues (Fig. 6). The entire tooth with

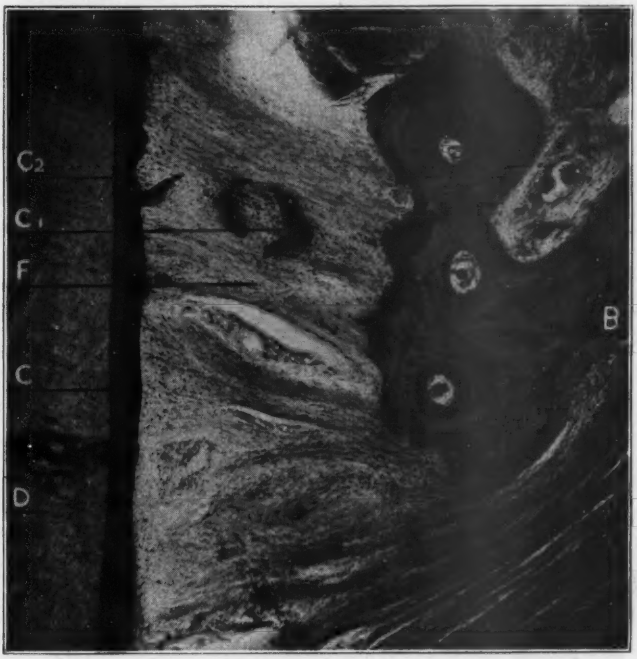

Fig. 7.—High magnification of the alveolar margin on the lingual side of the left second upper bicuspid (Fig. 4). *D*, dentin; *C*, cementum; *B*, alveolar bone; *F*, fibers stretched between bone and cementum; C_1, small splinters of cementum torn away from the tooth surface at C_2.

its alveolus, from the mechanical standpoint, can be compared with a pin in a tube. The pin can move only up and down, and the excursions of this movement, in our case, are determined by the arrangement and the resistance of the fiber bundles by means of which the tooth is suspended in its bony socket. The force that moved this tooth did have a slight lateral component, as is indicated by the fact that the buccal cusp is more worn than the lingual, which shows that

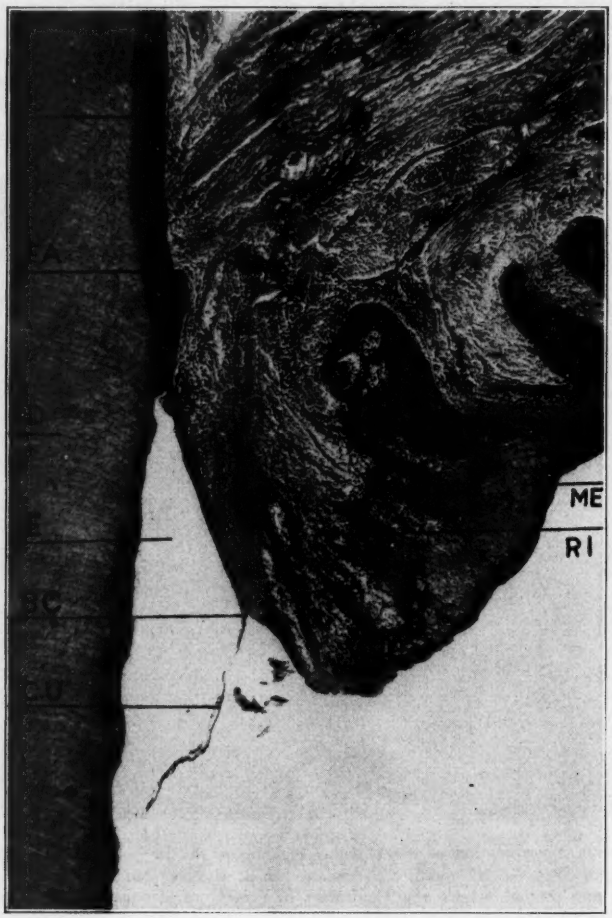

Fig. 8.—Gingival margin on the lingual side of the left upper second bicuspid (Fig. 4). *D*, dentin; *E*, enamel; *C*, cementum; *ME*, mouth epithelium; *BC*, bottom of the gingival crevice; *CU*, cuticle; *EA*, deepest point of the epithelial attachment on the cementum; *RI*, retro-epithelial round cell infiltration.

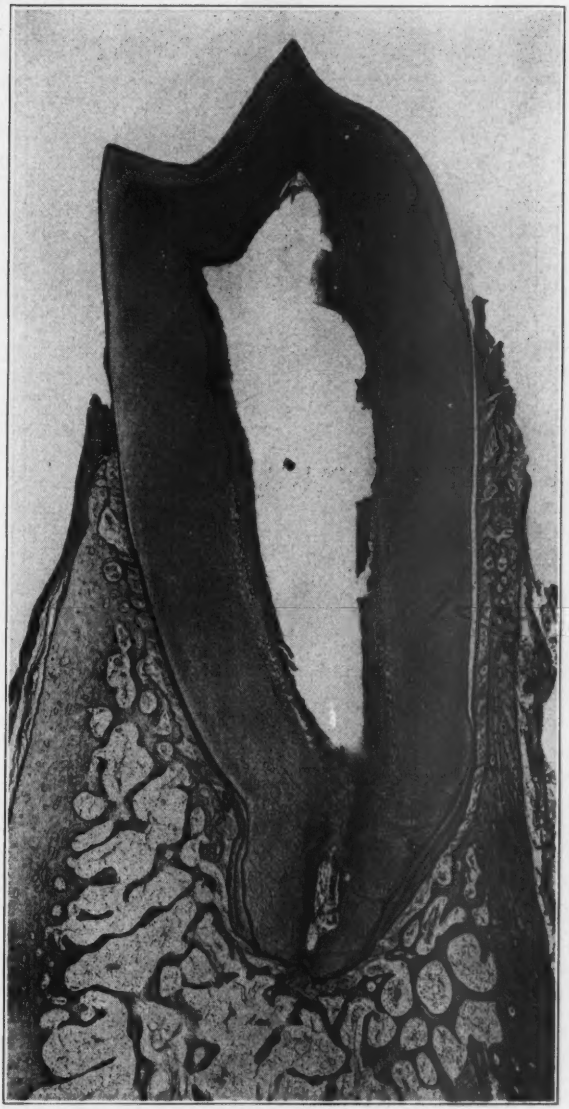

Fig. 9.—Lower left second bicuspid (Fig. 2, *B*); buccolingual section. There is a wide periodontal space and strong fiber bundles. The pulp was lost during sectioning.

Fig. 10.—Lower left first bicuspid (Fig. 2, *B*); buccolingual section. There is a narrow periodontal space, with only a few fiber bundles irregularly distributed. (The pulp was lost during sectioning.)

the tooth was slightly tipped toward the buccal side by the occlusion. In fact, when studying the entire series of sections, we could find an area on the lingual side near the alveolar margin where a very small piece of cementum had been torn away from the tooth surface and is lying free between the fibers (Fig. 7). The resulting defect in the cementum is partially repaired. Findings like this always indicate the action of a great force by which groups of fibers are torn loose. Sometimes, these fibers are so strongly attached that pieces of cementum tear away from the root with them.

Another important point in this connection is the condition about the gingival crevice of this tooth. On the labial side of the specimen, the epithelial attachment has been torn away from the tooth surface, and, therefore, the exact depth of the crevice cannot be measured. The pocket could not have been deeper than 0.1 or 0.2 mm. On the lingual side, the original relationship of the gingiva to the tooth is still intact. In a higher magnification of this area (Fig. 8), it can be seen that the bottom of the crevice is located on the enamel. Hornified cuticle indicates the point of detachment of the epithelium from the enamel. The deepest point of the epithelial attachment has passed the cemento-enamel junction and is found on the cementum (*EA*). The retro-epithelial connective tissue shows round-cell infiltration. The depth of this crevice is about 0.1 mm. or, practically, zero. It should be especially noticed that although this tooth was subjected to an unusual amount of functional stress, it is surrounded by healthy gums and has very shallow pockets.

Although it has been the general conception that occlusal trauma causes tissue destruction about the tooth, some careful observers have always emphasized the occurrence of overloaded teeth without the slightest evidence of damage. To quote MacMillan[4]: "Clinically, and from the study of specimens, it has been observed that a single remaining molar bearing all the masticatory activities of the teeth that have been extracted more often continues free from caries and alveolar border disturbances, when obviously the total sum of the occlusal stress is several times greater than when the other teeth were present. . . . When a tooth is under increased occlusal stress, the bone is physiologically adapted to take care of that increase. The point where physiologic stimulation ends and "traumatic occlusion" theoretically begins has not been shown."

Gottlieb, Orban and I made a number of experiments on dogs to study the influence of great occlusal stress on teeth. Gottlieb[5] has published some of these findings. No influence of the overstress on the gums or epithelial attachment could be found. The bone and periodontal membrane became adapted to the amount and to the direction of the force. The overloaded teeth of the jaw described in this paper show, on the whole, the same reaction to masticatory stress as that found in the experiments above. In the latter part of this paper, we shall see that the gums around the functionless teeth were inflamed and discharged pus, as these teeth lacked natural cleaning and, hence, they became covered with deposits that acted as a chronic irritant to the gums and caused filth pyorrhea.

Before proceeding to the description of other teeth of this jaw, it seems ad-

4. MacMillan, H. W.: Clinical Significance of Disuse Atrophy of the Alveolar Process, J. A. D. A., 14:697 (April) 1927.

5. Gottlieb, Bernhard: Paradentalpyorrhoe und Alveolaratrophie, Fortschr. d. Zahnheilk., 5: 401-414, 1928.

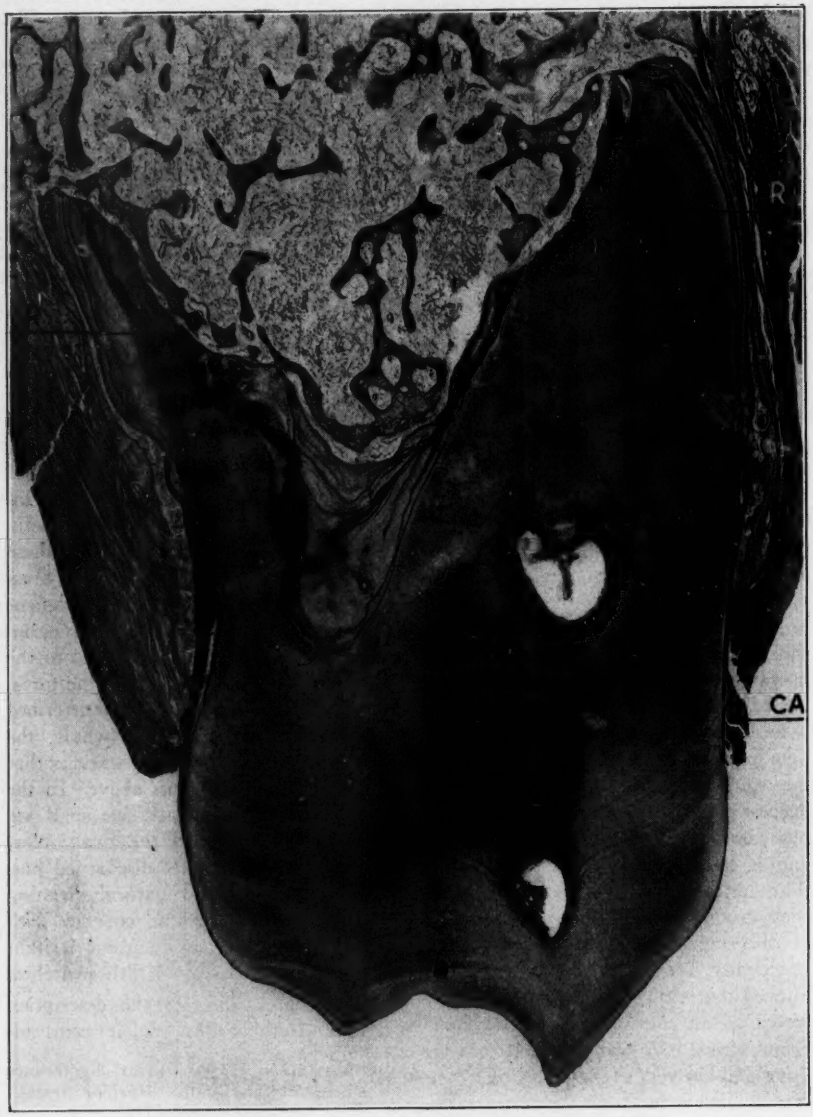

Fig. 11.—Left upper second molar; buccolingual section through the mesiobuccal and a part of the lingual root. *R, R,* areas of resorption of the roots; *CA,* calculus on the labial side of the tooth (compare Fig. 2, below).

Fig. 12.—Left upper second molar; buccolingual section through the lingual root. *R, R,* areas of resorption of the root; *P,* pulp canal; *CA,* calculus; *M,* large marrow spaces with few delicate bone trabeculae.

visable to consider not only the structures, but also the width of the periodontal membrane. Until a few years ago, only vague statements about the width of the periodontal membrane were given. No definite relations to the kind of tooth, age, function and other such factors were known. Kellner[5] was the first who stated that, in functioning teeth, the periodontal space is larger than in functionless teeth of the same jaw. Klein[6] made measurements on a large amount of material, with careful consideration of all circumstances that might be of importance to this question. Klein, too, found that the periodontal width depends on the function. His findings can be summarized by the words: the more function, the wider the periodontal membrane. The smallest figures in measuring periodontal spaces were found in retained (unerupted) teeth, these teeth being absolutely functionless. In the jaw that is the subject of this paper, measurements were made of the periodontal width in

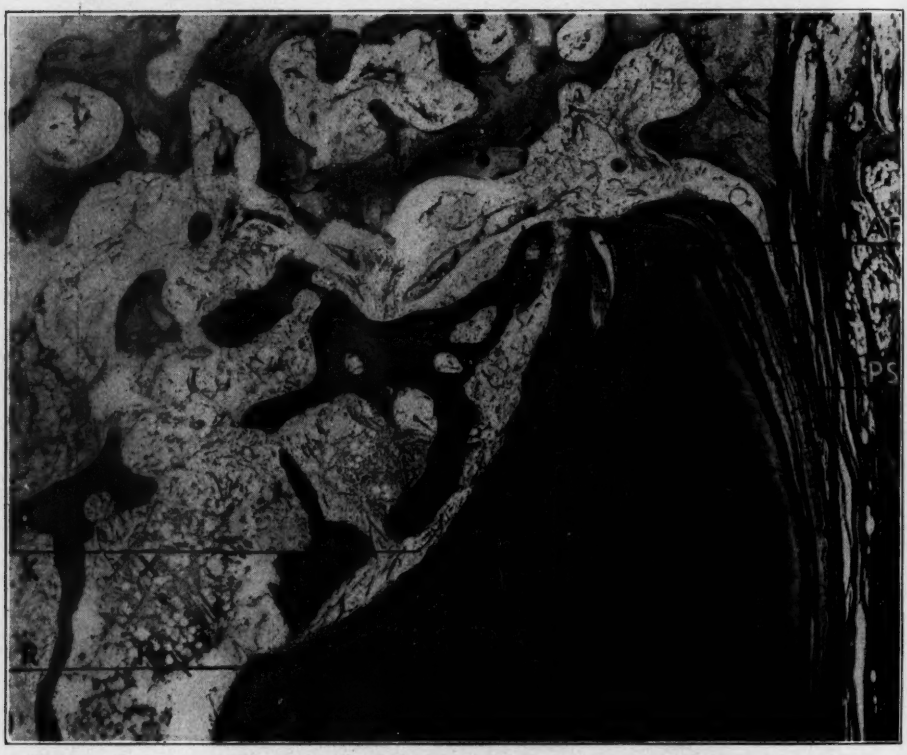

Fig. 13.—Higher magnification of the buccal apex in Figure 11. *AF*, apical foramen. From *AF* to *R*, the process of resorption has destroyed the original outline of the root. *PS*, narrow periodontal space on the buccal side of the root. At *X*, the periodontal space is large and irregular.

6. Klein, A.: Systematische Untersuchungen ueber die Periodontalbreite, Ztschr. f. Stomatol., 1928, No. 4.

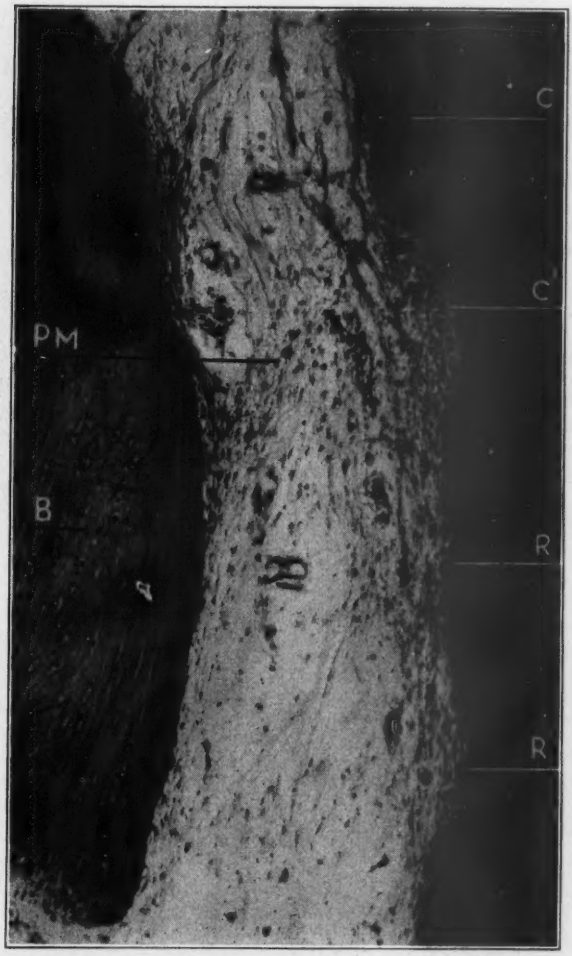

Fig. 14.—High magnification of area *X* in Figure 13. *C*, newly built cementum deposited on the resorbed dentin surface; *C'* beginning repair; *R*, resorption of the dentin by mononuclear osteoclasts; *PM*, loose fibrous tissue of the periodontal membrane containing round cells. The latter are a result of the septic condition of which the patient died. *B*, alveolar bone.

the way that it was done by Kellner and Klein. The results for the left upper second bicuspid were: average width of the periodontal space at the entrance of the alveolus, 0.35 mm.; average width of the periodontal space in the middle of the alveolus, 0.28 mm.; average width of the periodontal space at the fundus of the alveolus, 0.30 mm.

This distribution of the width of the

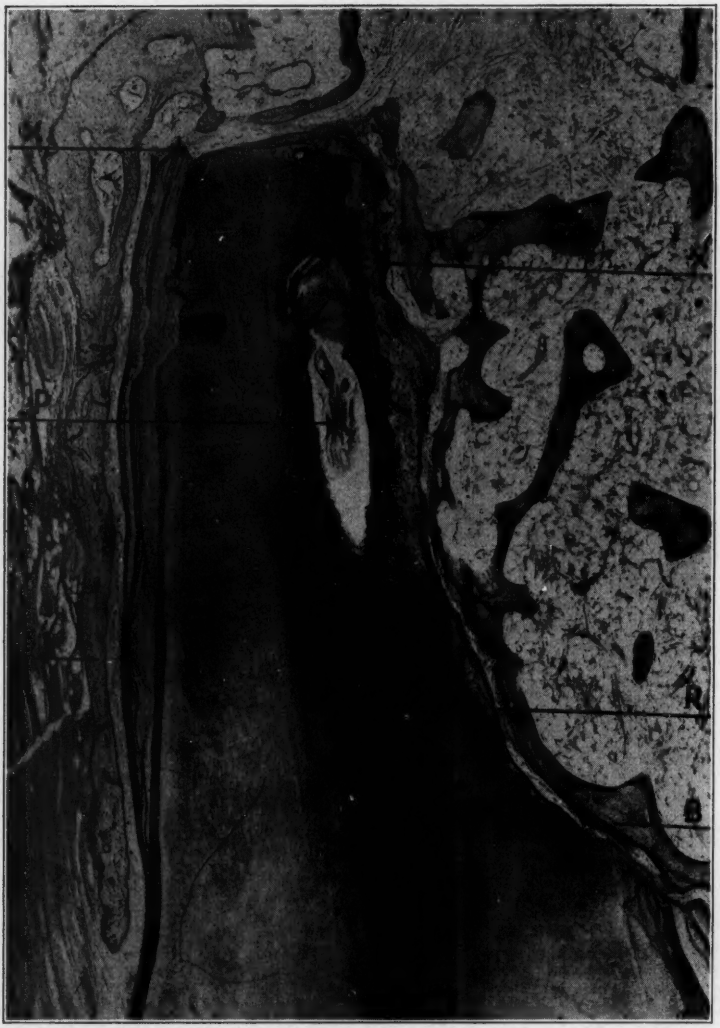

Fig. 15.—Higher magnification of the end of the lingual root (Fig. 12). Deep resorption on the side (*R*) and at the end (X^1) of the root. The alveolar bone (*B*) follows the outline of the resorption, forming a very narrow periodontal space. *B'*, buccal plate of the alveolus; *P*, pulp.

periodontal membrane; namely, greatest at the entrance of the alveolus, least in the middle of the alyeolus, and a little greater again around the apex, makes the shape of the periodontal space that of a double cone. This shape corresponds to

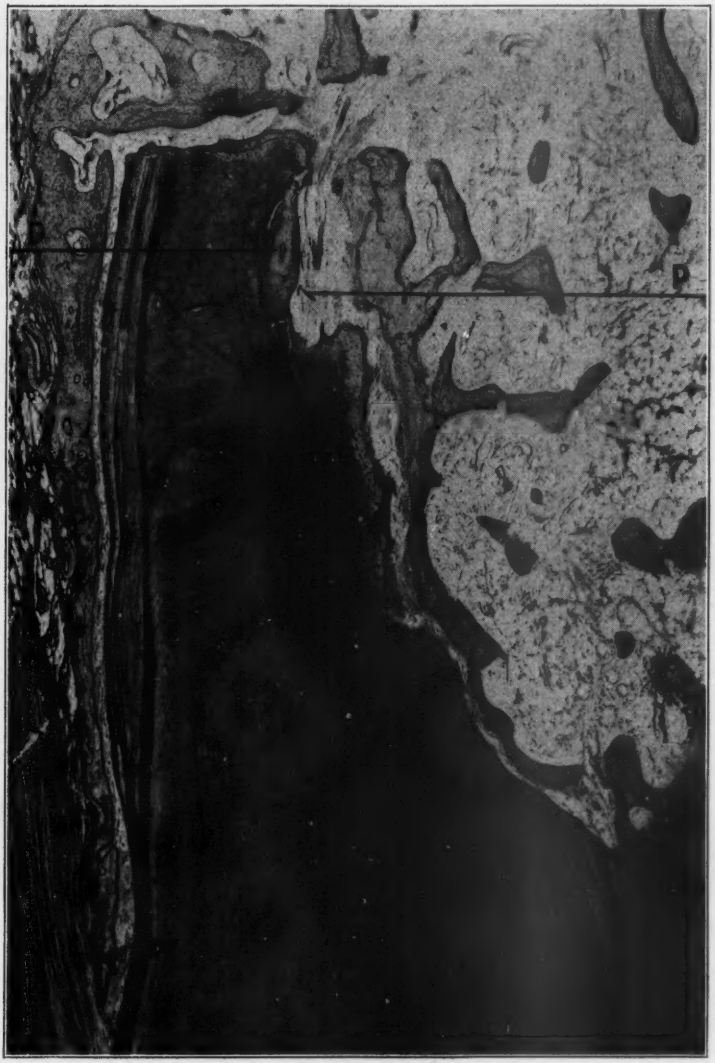

Fig. 16.—Another section through the same root end as shown in Figure 15. *P*, pulp strand passing through the apical foramen; *D*, denticle adherent to the wall of the former root canal.

the location of the fulcrum of the tooth, which is found at about the border between the middle and the apical third of the root. The root, therefore, in its lateral movements makes the greatest excursions at the alveolar margin, and

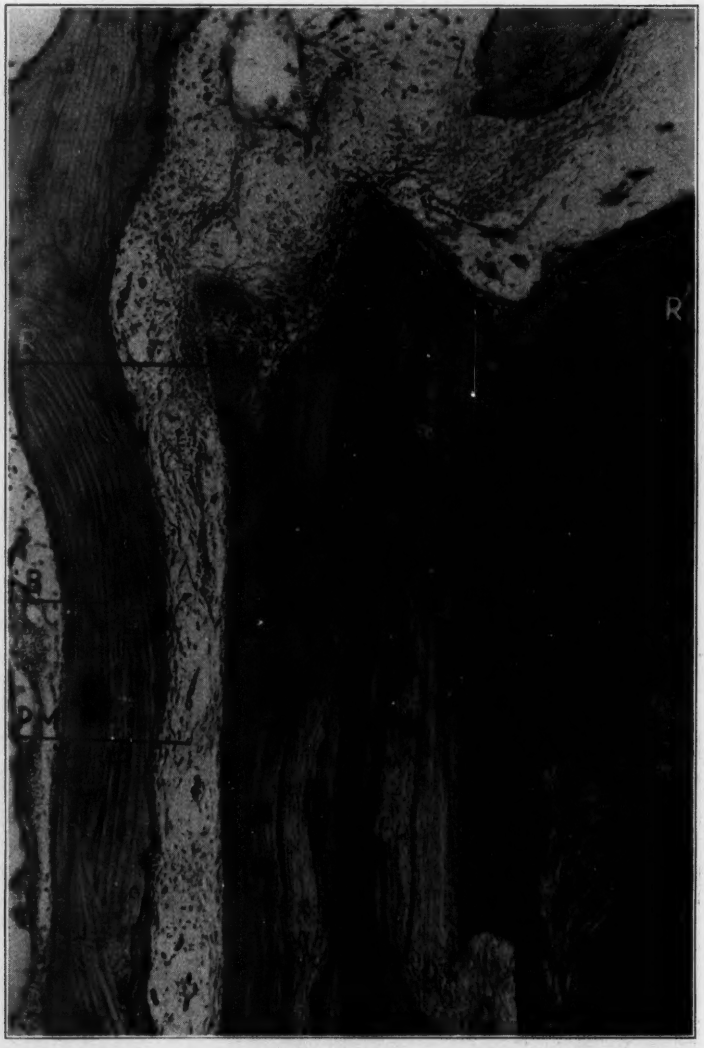

Fig. 17.—High magnification of area *X* in Figure 15. *R*, resorption of the original cementum on the root surface; *R'*, repaired resorption of the cementum; *PM*, periodontal membrane consisting of loose connective tissue without fiber bundles. Compare the width and the structure of this periodontal membrane with the one shown in Figure 5. *B*, alveolar bone.

slightly smaller excursions at the apex. In addition to the width of the periodontal space on the sides of the root, the distance between the apex and the fundus

of the alveolus has been measured. This distance varied whether it was measured in an area of bone resorption (Fig. 6 at *R*, where figures of 0.40-45 mm. were obtained), or in an area where the tissue was compressed and where, therefore, no resorption could take place (Fig. 6 at *CO*). Here, a width of only about 0.15 mm. was found.

In summarizing the findings on this upper bicuspid, we can say that this tooth was exposed to a great amount of functional stress in an almost vertical direction. The periodontal membrane became adapted to this surplus of work by a thickening and strengthening of its fiber bundles. The periodontal membrane is relatively wide, in order to allow the physiologic motility that is necessitated by the heavy function. No influence whatever of the increased function on the gingival margin could be found.

The next tooth to be described is the antagonist of this upper bicuspid, the lower left second bicuspid. This tooth showed similar conditions to those found in the antagonist (Fig. 9). A general view reveals dense compact bone around the tooth. The fiber bundles of the periodontal membrane are arranged in a way similar to those of the antagonistic tooth. They are very dense and regular all around the sides of the root and this arrangement can be followed down to the very apex. As has been mentioned before, this distribution of the fibers indicates the presence of an occlusal force, mainly in an axial (vertical) direction. On the labial side of this tooth, the gingiva was torn away during necropsy.

Fig. 18.—Root end of the upper left third molar. *R, R,* shallow areas of resorption at the root end; *B,* alveolar bone; *F,* fat marrow.

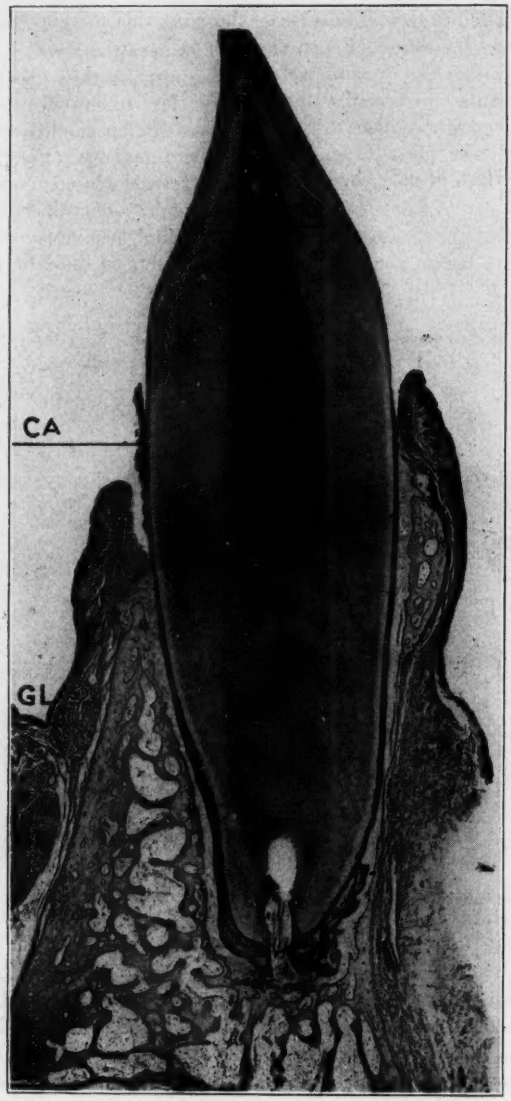

Fig. 19.—Left lower central incisor; labiolingual section. *CA*, calculus; *GL*, salivary glands on the inside of the mandible. The direction of the occlusal stress on the crown was toward the left of this picture. The direction in which the apex moved during function was toward the right.

On the lingual side, the soft tissues are in place and a shallow gingival crevice is to be seen on the cementum. The pulp of the tooth was lost in preparation.

The figures obtained by measuring the width of the periodontal membrane of this tooth were: average width at the entrance of the aveolus, 0.30 mm.; average width in the middle of the alveolus, 0.24 mm.; average width at the fundus of the alveolus, 0.20 mm.; distance of the apex from the fundus of the alveolus, 0.30 mm. In comparing the figures of the width of the periodontal membrane

Fig. 20.—Root end of the left lower central incisor (Fig. 19). *PM*, periodontal membrane, showing dense strong fiber bundles; *C*, regular layers of cementum on the lingual side of the root end; *HY*, irregular hypercementosis on the labial side of the root end.

of this lower bicuspid (0.30; 0.24; 0.20) with the ones obtained by measuring the upper bicuspid (0.35; 0.28; 0.30 mm.), we find a difference in the ratio between the different levels of the alveolus. In the upper bicuspid, the figures indicate a location of the fulcrum at a point a little above (toward the apex) the middle of the root. In the lower bicuspid, the figures gradually decrease from the entrance of the alveolus toward the apex. That indicates a gradual decrease of the excursions from the crown toward the apex. This difference between the upper and lower tooth may be explained by the conditions of the fibers at the apex. In the upper bicuspid, there were no apical fibers that could retain the tooth, and thus the apex could more easily make lateral excursions. In the lower tooth, the apex was held firmly in place, and the relatively small lateral component of the stress merely moved the crown. In addition to that, the apical part of the root of the lower bicuspid was curved and thus less likely to be moved laterally.

The next tooth to be described is the left lower first bicuspid. This tooth can be considered as a representative of group 2, that is, a tooth in slight function or only incidentally in function. As can be seen in Figure 2 at *B*, the left lower first bicuspid had no antagonist; but in studying the casts made from the jaws and put in an articulator, it became evident that, during the masticatory movement, this tooth came in contact with the upper second bicuspid. A general view of the tooth (Fig. 10), together with the surroundings in a buccolingual section, reveals that the periodontal space is narrower than in the neighboring tooth (compare Fig. 9). The fiber bundles are much weaker, and loose tissue fills the space between root surface and bone, especially in the apical half of the root. The figures obtained by measuring the periodontal width of this tooth are the following: average width at the entrance of the alveolus, 0.14 mm.; average width in the middle of the alveolus, 0.10 mm.; average width at the fundus of the alveolus, 0.12 mm. The distance between the apex and the fundus of the alveolus could not be determined, as no central sections through the very apex of this tooth could be obtained because of the curvature of the root.

In comparing these figures with the ones obtained from the neighboring second bicuspid, which was exposed to intensive function (first bicuspid: 0.14, 0.10, 0.12 mm.; second bicuspid: 0.30, 0.24, 0.20 mm.), we realize the important fact that the periodontal membrane is almost exactly twice as wide in the tooth with the intensive function. This fact is again confirmation for the results obtained by Kellner[3] and Klein[6] by their measurements. Both these authors stated that functioning teeth always have larger periodontal spaces than the functionless teeth of the same jaw. Gottlieb[7] describes a case in which an upper cuspid was lying unerupted in the jaw, and its root was in close proximity to the root of the erupted, normally functioning first bicuspid. Both roots were sectioned in one specimen, and the difference between the narrow periodontal space of the cuspid and the wider one of the bicuspid can be plainly seen in this photomicrograph. Klein, who took measurements from a large amount of material, found an average of 0.20 mm. for the functionless teeth and 0.24 mm. for functioning teeth. Of course, there are individual variations. In some jaws, the difference in the periodontal width between the two types of

7. Gottlieb, Bernhard: Paradentalpyorrhoe und Alveolaratrophie, Fortschr. d. Zahnheilk., 1929, No. 5 (Figs. 254 and 255).

Fig. 21.—Higher magnification of the lingual side of the root end. *C*, cementum; *F*, fiber bundles of the periodontal membrane; *B*, bundle bone; *P*, pulp.

teeth will be greater, in others, smaller, but the functioning tooth always shows the greater figures. In the case described here, this difference is an especially striking one, as will be seen in a study of the entirely functionless molars.

The three upper molars that were present in this jaw will be studied and described together, as all three are devoid of antagonists and therefore present the same functional conditions. The marked difference between these functionless molars and the functioning bicuspids is evident in each one of the teeth. A general view of the left upper second molar is shown in Figure 11. This section goes through the apex of the mesiobuccal root and through a part of the lingual root. Another section through the same tooth going through the lingual root close to the foramen is reproduced in Figure 12. As the three roots of this molar were curved and ran in different directions, it was not possible to obtain absolutely central sections. The study of the entire series made it possible to reconstruct fairly accurately the changes and peculiarities of this tooth. In all these sections, the pulp, which was normal and vital, was lost in preparation. The most striking fact about the roots of all the three molars is that they show extensive resorption of cementum and dentin. In Figure 11, on the buccal (right) root, there is resorption on the inner side extending over about one-half of the root length. In this section, the apical foramen may be seen and the bundle of blood vessels and nerves running through it. The lingual root in this section appears shortened by resorption. This becomes more evident in Figure 12, a section close to the foramen of the lingual root. The root looks as if it were cut off at right angles to the root canal. In the areas of root resorption, frail trabeculae of bone have again been built toward the root surface. This bone exactly follows the outline of the areas of resorption. The spongy bone above this tooth is greatly reduced; only a few frail trabeculae of bone are to be seen. The periodontal space is much narrower than in the bicuspids (Figs. 4 and 9). In the photographs of the jaw (Fig. 2), it has been shown that the labial side of the upper molars was covered with calculus. In the histologic sections, the calculus can be plainly seen (Figs. 11 and 12, *CA*). It covers a shallow pocket; the gingival tissues underneath show a dense infiltration, and round cells (pus) are discharged through breaks in the epithelium: the typical histologic picture of filth pyorrhea. On the lingual side, where there were not so many deposits, the gums look much healthier; almost no pockets are present. On the buccal as well as on the lingual side, the bottom of the gingival crevice has passed the cemento-enamel junction. A higher magnification of the apex of the buccal root in Figure 11 shows the deep resorption on the inner side of this root (Fig. 13). The surface of this area is almost entirely covered by a thin layer of new cementum. In some places, an active process of resorption is still going on. Figure 14, a high power photomicrograph of the area at *X*, Figure 13, reveals a typical condition as it is found all over the roots of the three functionless molars of this jaw. Resorption has taken place over all the dentin surface that is reproduced in this figure. At *C*, a layer of newly built cementum is deposited on the irregularly resorbed dentin. At *C'*, an initial stage of repair can be seen consisting of a delicate deposit of uncalcified cementum on the dentin. At *R*, there is active resorption of the dentin still going on. The resorbing cells are small, spindle-shaped and mononuclear. The periodontal membrane consists of a very loose tissue with irregular tender fibers. The round cells that can be seen in this tissue are found all over the tissues of this jaw and are due to the general septic condi-

Fig. 22.—High magnification of the labial side of the root end; irregular hypercementosis. *R,R,* former areas of resorption, now being repaired by new cementum; *F',* calcified insertion of a fiber bundle; *B,* alveolar bone; *P,* pulp; *L,* resorption of the lamellated bone.

tion of which the patient died. This infiltration of the jaw tissues in cases of sepsis has lately been described by Schoenbauer.[8]

The fact that active resorption still takes place on these teeth is of greatest importance. If we found merely repaired areas of resorption, we could not be sure whether we are dealing with the result of former conditions, perhaps of a trauma that existed at the time when this tooth still had antagonists. We are able to show the presence of active resorption on all these functionless teeth, but no resorption of tooth substance whatever on the functioning ones.

The complete absence of typical fibrous periodontal membrane in this tooth can be very plainly seen in Figures 14 and 17. The periodontal space is filled with a loose connective tissue, and delicate blood vessels are running in all directions through it.

In Figure 15, a higher magnification of the lingual root in Figure 12, the original outline of this root ends at *X*. The entire apical part from *X* toward the apex has been destroyed by the process of resorption. The exposed dentin surface has been covered by a new thin layer of cementum, and a new periodontal space has been restored. The thin lamina of alveolar bone (*B*) closely follows the outline of the tooth surface, leaving only a very narrow periodontal space. Loose connective tissue fills this space, and only a few fibers are present, most of them running parallel to the tooth surface. At *X* and *X'*, active resorption is still going on. At *X*, the original cementum is resorbed. At *X'*, the newly deposited cementum is again attacked and eliminated by resorption. A few sections further on in the series, we find the apical foramen of this root, namely, the place where the blood vessels and nerves enter the root canal after the original root end has been lost (Fig. 16). An adherent denticle, *D*, that originally was located in the root canal is now exposed by the process of resorption and is found forming one side of the foramen.

Both Figure 15 and Figure 16 show the delicate, atrophic structure of the alveolar bone, due to the lack of an adequate functional stimulus. In Figure 17, a higher magnification of area *X* in Figure 15, resorption of the original cementum that covers the root surface can be seen. Close to it, there is another area of resorption that is already repaired. The periodontal membrane consists of loose fibers running parallel to the tooth surface, just as it has been described by Kellner.[4]

It is of great interest to compare the measurements of the periodontal width of this functionless left upper second molar with the figures obtained from the functioning second upper bicuspid of the same side (Fig. 2). The figures for the periodontal width of the left upper second molar were: average width at the entrance of the alveolus, 0.13 mm.; average width at the middle of the alveolus, 0.10 mm.; average width at the fundus of the alveolus, 0.8 mm.; distance of the root end from the fundus of the alveolus, 0.10 mm. In comparing these figures with the ones obtained from the upper second bicuspid (0.35, 0.28, 0.30 mm.), it becomes evident that the periodontal width of the functionless upper molar is about one-third the width of the heavily functioning second bicuspid of the same side of the maxilla.

The periodontal membrane of the right upper second molar (Fig. 2) showed the same structure as the one just described.

8. Schoenbauer, F.: Histologische Befund im Kiefer bei Sepsis und Leukaemie, Ztschr. f. Stomatol., 1929, No. 11.

Resorption had occurred over a large part of the root surface. Part of the resorbed areas were repaired, but, in some places, resorption was still going on. The roots of this tooth were so curved and bulky that it was not possible to obtain central sections. The few areas that could be measured showed a periodontal width between 0.10 and 0.14 mm., these corresponding to the figures obtained from the similar tooth on the other side of the maxilla.

The left upper third molar shows the smallest figures in the measurements of the periodontal width: average width of the periodontal space at entrance of alveolus, 0.10 mm.; average width of periodontal space at middle of alveolus, 0.06 mm.; average width of periodontal space at fundus of alveolus, 0.06 mm.; distance of root end from fundus of alveolus, 0.05 mm. A photomicrograph of the root end (Fig. 18) of this tooth reveals the characteristics of the functionless teeth in this jaw: very narrow periodontal space; resorption at the flattened root end, most of which is repaired; frail structure of the spongy bone; on top of the root end, delicate trabeculae of bone following closely the outline of the root surface.

There is still another point that should be mentioned in this connection. Kellner measured the thickness of the cementum on corresponding teeth of the same jaw, some being in function and others functionless. He found that the functionless teeth had a thicker cementum than the corresponding functioning teeth of the other side. Thus, the thickness of the cementum showed a reverse ratio, in its relationship to function, to that of the periodontal width. In the case described here, the width of the cementum could not be used for our study of the influence of function, as it was not possible to compare corresponding teeth. A comparison of different kinds of teeth, for instance, bicuspids and molars, is not possible on account of the anatomic difference in thickness and arrangement of the cementum. Thus, only the periodontal width could be measured.

Finally, it will be of interest to hear some details about the anterior teeth in this jaw. (Fig. 1.) The two upper central incisors could not be compared with each other, as, in the right one, there was too much destruction of the periodontal tissues due to the presence of an extensive periapical inflammation. The figures for the width of the periodontal space of the left upper central incisor were: average width at the entrance of the alveolus, 0.28 mm.; average width at the middle of the alveolus, 0.15 mm.; average width at the fundus of the alveolus, 0.24 mm. These figures, as compared with the ones obtained from the bicuspids, would indicate heavy function. The main point of interest is the fact that the middle of the alveolus is relatively narrow, comparatively much narrower than in the bicuspids. An explanation of this is found by studying the occlusion of this central incisor (Fig. 1). This tooth was exposed to an almost strictly horizontal stress (lateral), causing a tipping of the crown. This type of movement caused relatively wide excursions at the alveolar margin, and less extensive movements at the apex. In the middle of the alveolus, corresponding to the location of the fulcrum, there is a very narrow periodontal space. In the bicuspids, where the direction of the stress was more nearly vertical, the distribution of the periodontal width over the alveolus is more regular.

This explanation of the decrease of the periodontal width in the middle of the alveolus becomes more evident in a comparison of the two lower central in-

cisors. The right lower central incisor (Fig. 1) had a slight end-to-end contact with the antagonistic gangrenous root of the right upper central incisor. This contact evidently caused a moderate pressure into the alveolus (vertical pressure). The left lower central incisor touched the antagonistic upper incisor on the lingual plane of the latter and thus was tipped lingually by a horizontal force. A general view of this tooth (Fig. 19) reveals the presence of moderate calculus and inflammation of the gums on the lingual side. The periodontal space is fairly large, and, at the root end, we see a considerable amount of hypercementosis, as the result of a functional stimulus. The comparative measurements of the two lower central incisors are:

Right lower central incisor: slight function in the direction of the axis of the tooth (vertical direction): average width of the periodontal membrane at the entrance of the alveolus, 0.18 mm.; average width of the periodontal membrane at the middle of the alveolus, 0.16 mm.; average width of the periodontal membrane at the fundus of the alveolus, 0.17 mm. The smallest figure for the periodontal width of this tooth that could be found was 0.08 mm. This point was located below the middle of the root, a little toward the apex.

Left lower central incisor: occlusal stress in a horizontal direction (lingual tipping of the crown): average width at the entrance of the alveolus, 0.26 mm.; average width at the middle of the alveolus, 0.10 mm.; average width at the fundus of the alveolus, 0.24 mm.

From this comparison, two facts are evident:

1. The right lower central incisor, in accordance with the smaller amount of function shows, on the whole, a narrower periodontal space.

2. The width of the periodontal space of the right lower central incisor, which was exposed to a slight vertical pressure, is about uniform all over the root surface. The measurements of the left lower incisor, which was exposed to a strong horizontal stress (tipping force) show the minimum width at the middle of the alveolus, and considerable increase of the width toward the upper and lower end of the root, where the width is more than double that in the middle.

The latter fact illustrates plainly the location of the fulcrum of a moving tooth. It shows that the statement of A. M. Schwarz[9] that the fulcrum of a tipping single rooted tooth is in the middle of the alveolus or a little way toward the apex is correct, and that Häupl and Lang[10] are wrong when they claim that the fulcrum is near the alveolar margin.

A higher magnification of the apical end of the left lower central incisor (Fig. 20) shows a well-developed fibrous periodontal membrane and an irregular hypercementosis, evidently due to calcification at the insertion of Sharpey's fiber bundles. This type of hypercementosis has been determined by Gottlieb as the reaction of a sound, highly functioning tooth to occlusal stress of long standing. Gottlieb showed that an excess of stress may cause slight resorption of the tooth surface, and that these areas of resorption in a well-reacting tooth are readily repaired by newly built cementum. In other places, this new formation of cementum takes place without preceding resorption, and thus a hypercementosis

9. Schwarz, A. M.: Ueber die Bewegung belasteter Zaehne, Ztschr. f. Stomatol., 1928, No. 1. Mathematische Ueberpruefung des Verhaltens belasteter Zaehne, ibid., 1928, No. 4.

10. Häupl and Lang: Marginale Paradentitis, Berlin, 1927.

develops. Gottlieb showed how the direction of the force can be determined by the localization of this hypercementosis. In this lower incisor with its marked tipping movement, we find an irregular jagged hypercementosis merely on the right (labial) side of the apex.

On the lingual side (Fig. 21), there is a gradual increase of the thickness toward the apex by a regular deposit of parallel layers of cementum. The presence of bundle bone (*B*) indicates that the tooth has moved away from this area. A higher magnification of the hypercementosis on the labial side (Fig. 22) reveals the presence of many old areas of resorption (*R-R*) in the cementum that are completely repaired. Irregular new cementum has been deposited, and thus the surface of the root is jagged. The investigations of Gottlieb give us the clew for these findings. The right side of the apex in Figures 19 and 20 is the side of pressure. This becomes evident by the direction in which the crown was tipped (see arrows in Figure 19). (As the fulcrum is located about in the middle of the tooth, the apex necessarily made excursions in the opposite direction; namely, toward the labial side.) By this pressure, the alveolar bone as well as the root surface to a small extent became resorbed. As the tooth belonged to the type that Gottlieb calls well-reacting, these areas of resorption were readily repaired, and thus the hyperplasia on the side of pressure developed.

SUMMARY

The jaws of a man, aged 38, were sectioned and studied. Varying functional conditions were found on the different teeth according to the presence or absence of antagonists. Histologically, it could be found that the periodontal membrane of the functioning teeth consisted of dense, strong fiber bundles. Thus, these teeth became sufficiently adapted to a great amount of stress. The distribution of the fibers on the tooth surface corresponded to the direction of the force which could be determined by studying the occlusion. No direct influence whatever of the function on the gingival margin could be seen. The functionless teeth showed poorly developed periodontal membrane, consisting merely of loose connective tissue almost without fiber bundles. On the roots of the entirely functionless teeth, there were large areas of resorption that were partially repaired. In other places, an active process of resorption was still going on.

A comparison of the periodontal width of functioning and functionless teeth, as shown in the accompanying table, substantiates the measurements of Kellner and Klein.

COMPARISON OF PERIODONTAL WIDTH OF FUNCTIONING AND FUNCTIONLESS TEETH

	Heavy Function Left Upper 2nd Bicuspid mm.	Light Function Left Lower 1st Bicuspid mm.	Functionless Left Upper 3rd Molar mm.
Average width of periodontal space at entrance of alveolus	0.35	0.14	0.10
Average width of periodontal space at middle of alveolus	0.28	0.10	0.06
Average width of periodontal space at fundus of alveolus	0.30	0.12	0.06

CONCLUSION

In the human jaw described in this paper, heavy function acted as a stimulus for the periodontal tissues, which became hypertrophic. Thus, the tooth became adapted to stand the increased stress without damage. On the other hand, lack of function caused an involution of the periodontal soft tissues and atrophy of the bone.

These findings indicate that we are mistaken if we draw any general conclusion concerning the influence of overfunction on the teeth. We have always to consider the way in which the periodontal tissues are able to react to this increased stimulus. The jaw described above belongs to the type that Gottlieb calls "well-reacting." Therefore, the heavy excess stress on some of the teeth did not cause damage. The same or perhaps a smaller amount of stress exerted on "poorly reacting" teeth might have brought about severe damage and loosening of the teeth. It is not the amount of stress or function that is responsible for the final outcome and fate of the teeth, but the way in which these teeth and their surroundings react to such stress.

DISCUSSION

E. E. Monson, Salt Lake City, Utah: Often, the roentgenogram shows that the bone underneath the tipped tooth is very porous. Is this due to the fact that the traumatic occlusion, because of the tipping of the tooth, has caused the bone to recede under the tooth, or has its lack of traumatism, its lack of use on account of being tipped, caused the recession of the bone under the tooth?

Rudolf Kronfeld, Chicago, Ill.: Either condition is possible. We have a tipping action that is so sharply directed toward one side that the other side of the tooth is entirely relieved, in which case we may expect atrophy of the bone, or the entire tooth is so much out of function that general atrophy of the bone ensues. We find distribution of the dense and the porous bone of the tooth under any functional condition that we are able to trace. Why? The tooth was exposed to stress in certain directions so that it received overstress in one direction and atrophy of the bone resulted on the opposite side.

I. A. Epstein, St. Paul, Minn.: This is one of the most practical contributions that I have heard from the Vienna school. The tissues in the jaws supporting the teeth are connective tissue, in type very similar to those we find in the blood-forming organs, with which I am more familiar. The reactions are identical. We have, as the result of increased functional demand, a stimulated activity; where we have a lack of function, we find the opposite. We also find a point at which exhaustion occurs, and I believe the conditions in the two cases are identical, since they are fundamental in the same types of tissue.

Hermann Becks, San Francisco, Calif.: I am not of the Austrian school even though I have a European accent. It was wise of Dr. Kronfeld not to draw far-reaching conclusions from the measurements of the peridental membrane. When we are dealing with an advanced case of "paradentosis"—the new term for pyorrhea employed in Europe—the measurements of the peridental membrane might have no special importance. I regret that the slides which were shown were not central but transverse sections, and I hope measurements of the peridental membrane were not made on them. It is known through the work of Bauer and Lang from the Innsbruck school that such measurements are of no value. On the other hand, Dr. Kronfeld showed a picture of a condition similar to osteoporosis on the second molar. This bone disease can be found in cases of paradentosis. In certain types of "genuine paradentosis," we are dealing with a resorptive process of bone on the basis of a systemic disease, as you may observe in the scientific exhibit hall. Through this resorptive process of bone, the width of the peridental membrane is extended so that no conclusions can be drawn from such measurements. We can produce these changes by diet in very young dogs. If we put animals (dogs) on a normal salt mixture or a low calcium salt mixture with or without cod-liver oil, we get an activation of osteoclasts and, through this, a resorption of the supporting bone of the teeth at an early age. Control roentgenograms show distinct differences. If I remember correctly, in the picture of the cuspid, there was an increased resorp-

tion of the alveolar bone, and a widening of the marrow space. This marrow tissue was not normal, as far as I could determine from the picture. It was changed into fibrous tissue, i. e., a pathologic condition, perhaps due to the overstress or "traumatic occlusion," as you might call it. The bone surrounding the second molar showed fat marrow, such as we find in adults or as an expression of the result of the disuse of bone. The fact that this molar has no opponent explains this picture very easily. In other words, we are dealing here with two different pathologic conditions, the one due to overstress and the other one due to lack of occlusion.

Dr. Kronfeld: We are not dealing here with an advanced case of "paradentosis," as I shall explain. I do not like the use of the word "pyorrhea." It implies such widely different conditions that there should be a clear understanding among the profession regarding it. One man calls a condition in which there is a little deposit of calculus on the teeth pyorrhea. He scales the teeth and the pyorrhea is gone. In another case, there is a loosening or drifting of the teeth; there is a rapid atrophy of the alveolar bone, but no calculus is present in the mouth; the mouth has always been well-kept and clean. This condition is also called pyorrhea. Someone else finds, in some older patients, that the root surfaces at the necks of the teeth are exposed, the mouth being otherwise in good condition. He calls that "paradentosis," merely because the roots are exposed, though the teeth are firm, the pockets shallow and the gums healthy. We do not get very far with this terminology, not much farther than we do with the word "pyorrhea." It is for this reason that the Vienna school, which has been mentioned here, always attempts to get a differential diagnosis as far as three main points are concerned. There are: (1) cases resulting from lack of proper care and of scaling and that improve readily after scaling has been done; (2) cases that are due to some inner disturbance of different origin in which the teeth start to loosen and to drift apart and to rotate or overlap—all conditions that characterize diffuse atrophy and that have nothing to do with local conditions or calculus and cannot be influenced by scaling; (3) cases in which parts of the roots are exposed and which are considered to be the result of a normal process of aging because it is found everywhere in older individuals, in man as well as in animals, and has nothing to do with pyorrhea. Dr. Becks says that we are dealing with a case of paradentosis. I should like to ask why he reaches this conclusion.

Dr. Becks: These two teeth represent typical pictures of paradentosis. I do not wish particularly to insist on the term paradentosis, but I apply it to all cases which show chronic bone changes, because I think that it is the best term we have at the present time to replace "pyorrhea." "Para" means around, "dens" the tooth, and "osis" expresses more or less the constitutional—trophoneurotic —or, as in these cases, the "functional" disturbance; in other words, a "chronic process around the tooth." This term given by Weski (1921) means more than "pyorrhea." I did not use the word pyorrhea, i.e., pus discharge out of the pocket. I cannot see that this condition is shown in these pictures. But we can see in the section of the molar that the position of the tooth has been changed and that there are calculus, pathologic bone changes, a chronic irritation of the gum tissue, a change in the width of the peridental membrane and a formation of secondary cementum. This means that we are dealing with a chronic pathologic process around the tooth on the basis of a functional disturbance, or "paradentosis." In the picture of the front tooth, we also saw a typical pathologic change of the alveolar process, a pathologic change of the marrow space, fibrous marrow tissue instead of fat marrow, i.e., bone which is not normal but pathologic. We saw changes in the width of the peridental membrane, and a slight inflammation of the gum tissue, although perhaps this is not severe enough to be called "pyorrhea." Since the four tissues—epithelium, cementum, bone and peridental membrane—are involved in the cases demonstrated by Dr. Kronfeld, I would call it "paradentosis" according to its definition by Weski.

Dr. Kronfeld: In Figure 2 (molar) is a condition that might readily be called "pyorrhea" because there is much calculus and consequently an inflammation of the gums. I do not hesitate to call this condition pyorrhea and to classify it as the first type, the type that disappears readily after scaling, and that, therefore, has been called filth pyorrhea. The pockets were very shallow; they had practically no depth, and the calculus extended up to the gums, which were inflamed.

As soon as the tooth is scaled, and the patient is shown the proper use of the toothbrush, this type of pyorrhea is cured. An entirely different thing is the bone changes, which have nothing to do with calculus. If, in this case, we construct a lower partial denture that puts this tooth back in function, then, I am sure, the bone will be rebuilt and reinforced. The bone is atrophic through lack of function and will become normal when the occlusal conditions are changed. If we scale, we might merely get a little better condition of the gums, but we cannot influence the bone. These things are entirely separate. If we bring the tooth into function reinforcement of the alveolar bone results. As far as the upper bicuspid is concerned (Fig. 4), I consider this bone ideal. I cannot imagine any better conditions than these found histologically in this tooth. Dr. Becks said that hypertrophy of the periodontal tissues and increased thickness of the bone are pathologic. This thickening of the bone is no "pyorrhea" and no "paradentosis." It cannot be compared with the inflammatory thickening around an apex with an infected root canal. In our case, we are dealing merely with a healthy functional hypertrophy comparable to the strong and hypertrophic bone and muscles of a well trained athlete. As far as the width of the periodontal membrane is concerned, Dr. Becks made a slight error, because the Innsbruck School, which he quoted, was the first to use extremely tangential sections from which to draw conclusions about "width."

Dr. Becks: I know there is a difference of opinion between the schools in Austria and Germany, and therefore I said at the beginning that I did not belong to the Austrian school. I am aware of the discussion between the Innsbruck school and the Vienna school in regard to the measurements of the peridental membrane. In answer to Dr. Kronfeld's correction, I know that the Innsbruck school made measurements, the accuracy of which were questioned, and I believe that Dr. Kronfeld misunderstood my statements. The points that I wish to stress are that there were no central sections shown during the lecture when the width of the peridental membrane was discussed. If we have abnormal apposition of bone or cementum, even though it is the expression of an ideal repair, I must regard this process as pathologic. Several weeks ago, a dentist found sclerotic bone around the apex of one of my teeth. He immediately recommended extraction on account of the "pathologic condition," but since I had had no trouble with it in the fifteen years since the root canal was filled, I explained that even though pathologic, it was a case of ideal repair, and the tooth should not be extracted. Even if we are dealing, in this case demonstrated by Dr. Kronfeld, with "ideal repair" by bone and cementum, we must, from a scientific standpoint and from judgment of all the other changes in the peridental membrane and epithelium, call this process "pathologic." It is a case of "paradentosis."

Dr. Kronfeld: Of course, if you make a section tangential enough, you can measure everything from that. It is evident that you need central sections, as Dr. Becks suggests. As far as my measurements here are concerned, they are taken from one single jaw and, of course, I wouldn't say that these measurements have general significance. But if Dr. Becks will take the time to measure a few hundred teeth, as it has been done, he will find these ratios are always the same. One will not always get the same numbers—one tooth might have a periodontal width of 0.30 mm., and another of 0.40. These are individual variations. But the ratio will always be the same. The tooth will be widest at the entrance of the alveolus, narrowest at the middle and a little wider at the fundus; and there are always wider measurements for functioning teeth and narrower for the nonfunctioning ones. In addition to these slides, I have the complete set of sections with me, from one side of the block, through the middle of the tooth to the other side of the block. I shall be glad to show all of them under the microscope, leaving it to the judgment of the audience whether the sections can be called central.[11]

11. Since the reading of this article, a paper by Kellner has appeared that deals with the subject of the periodontal width and breadth of the cementum (E. Kellner, Das Verhältnis der Zement—und Periodontalbreiten zur funktionellen Beanspruchung der Zähne, Zeitschrift f. Stomatol., January, 1931, pp. 44-62). From a study of the large amount of his material, this author comes to the same conclusions concerning the width of the periodontal membrane as those given in this paper; i.e., functioning teeth: thick periodontal membrane; functionless teeth: thin periodontal membrane.

SILICATE FILLINGS: THEIR USEFULNESS AND INDICATIONS*

By ROY JAMES RINEHART, D.D.S., F.A.C.D., Kansas City, Mo.

THE use of the silicate cement as a filling material has not as yet passed the experimental stage. Nevertheless, it is with us as one of the filling materials in general use. To quite an extent, it has supplanted the use of gold foil in the anterior teeth. Although insertion of the silicate cement as done by the majority may have been an abortion of good dentistry, this fact does not warrant its condemnation as a filling material.

GENERAL USE

As the moderate success of the silicate filling assures its continuous use, manufacturers and dentists must strive for its improvement with the objective that it may have complete recommendation as a restoration material. It is subject to as gross abuse as amalgam, but no more so. When it is used according to the instructions of manufacturers and with full understanding of all its peculiarities, the immediate results are delightful, and, in many instances, the results are excellent over a period of years; but it cannot be recommended to patients as a permanent filling: it is not in the same class with gold foil.

In writing on a subject of this kind, personal experience is the only information worth while. I, therefore, contribute what I have learned from the moderate use of the silicates since 1905. When Ascher's artificial enamel was introduced, I cautiously began its use. I tried this material in all classes of cavities and, as some of you will remember, the immediate results were excellent, but later there was extreme discoloration. The material was hard and lasting, some fillings holding their surfaces very well, while others were not so good.

One of the first fillings that I inserted was for my wife, restoring the mesial and occlusal surfaces of a lower first bicuspid in a deep seated cavity. The filling is giving excellent service today. The surface is hard and glasslike in appearance and it has always maintained contact.

SUCCESSFUL OPERATIONS

With such success in a few cases, we are led to believe that, even with the old Ascher product, there was at least an exact mixing and insertion procedure that produced a first-class result. This filling represented one of a goodly number of successes that I had with this product, and I have seen others. The unfortunate condition is that, while there was one of these successful fillings, there were perhaps at least twenty-five or more others not so successful or that failed to preserve the form of the tooth and, in many instances, caused the loss of vitality or loss of the tooth itself.

*Read before the Section on Operative Dentistry, Materia Medica and Therapeutics at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 23, 1930.

As every one is familiar with the products that later appeared on the market, there is little use in reciting the steps in technic except to emphasize here and there those procedures which, if carried out in detail, assure the best results. The powder and liquid must not be contaminated and the stoppers must be returned to the bottles immediately after removal of the portion needed. Surplus material from the slab must not be returned to the bottle. Agate instruments are indicated to avoid contamination affecting color. Metal instruments leave a certain portion of the metal in the material, thereby spoiling the refined product. The fingers should never touch the mix. The glass slab and instruments should be cooled. Breezes from a fan or from the outside should be avoided. Instructions for mixing and shading should be carefully carried out, each product having a mix peculiar to itself.

CAVITY PREPARATION

Outline form in approximal cavities should be well extended onto the labial and lingual surfaces for best results. Undercuts for anchorage are absolutely necessary, as silicates have little adhesion. In preparing cavities for the reception of silicate cement, the operator should use every precaution to protect the pulp from close approximation to the filling. This may be done by attention to the labiolingual convexity of the axial wall and, in the case of deep seated cavities, by placing a cement base or applying soft oxyphosphate zinc cement first and then adding the silicates. Cavity lining should be used when oxyphosphate zinc cement is not indicated.

Cavity linings or cement should not engage the margins of the cavity. These margins should be finished with a chisel maintaining square angles, as beveling is never indicated, for the reason that the thin margins on a beveled surface will check away leaving marginal defects. In the past, there have been many devitalized pulps under silicate cement fillings. This has been due to the free acid which remains under the filling and which is absorbed through the dental tubuli into the pulp, causing a constant irritation until devitalization takes place. I do not believe this danger has been removed from the product. In mixing, best results are obtained by preparing a third more material than will be used in the cavity. Powder and liquid should be placed on a glass slab and the powder drawn into the liquid, which is stirred gently until the powder disappears in the liquid. Enough powder should be incorporated until the mix is putty-like.

It is just as detrimental to have the mass too thick as too thin, and experience is the only teacher after one has become thoroughly familiar with the instructions which accompany each package of silicate cement. A thin celluloid strip should be placed between the teeth, the space being free and clean. The first cement applied to the cavity should be in a small amount, pressed in from the most accessible part of the cavity, the material being carried over the entire base of the cavity. This is followed by a second application. We should be sure that no air chambers have been created under the filling, and when the cavity is filled to overflowing, the strip is pulled over the soft mass, tightly compressing it and held firmly until the material has set sufficiently hard that distortion of the margins is impossible. The strip will immediately come loose from the filling. Cocoa butter is not necessary as a lubricant on the strip and its use often interferes with the hardness of the surface of the filling. A sharp chisel should be used to take away the excess or overhang on the enamel. If the strip is

properly handled, little finishing will be necessary. Finishing is more safely accomplished at a subsequent appointment.

My experience has been that it is better to flow paraffin or wax on the exposed surface of the filling than varnish. During the early use of this material in my practice, I religiously left the rubber dam on for thirty minutes, allowing the patient to sit in the chair or retire to the reception room until the time had elapsed. Later in my experience, I coated the fillings immediately with the varnish which accompanies the package. This was my undoing in my attempts toward the most successful use of the silicate cements. In most cases in which varnish was used and the patient was dismissed immediately, the fillings were rough within a few months.

RUBBER DAM A NECESSITY

The fillings inserted under the rubber dam, immediately covered with paraffin, and the rubber dam, left on for thirty minutes, have been an outstanding success in silicate restorations. Many of them have the same smooth surface that they had when first inserted and many of these fillings have been in more than ten years. This includes some two surface fillings. The filling in my wife's mouth was inserted in 1907.

FINISHING

Short cut methods using varnish as a cover I firmly believe are inimical to the successful use of this material. I believe that the varnish should be used in the cavity before filling, but the margins should be free from varnish. Finishing is accomplished by the use of linen strips, fine sandpaper disks and cocoa butter. The glazed surface left by the strip should be retained as nearly as possible, especially the contact point. Linen strips, with cocoa butter, are used to polish the cervical margins, plain linen strips with the silicate powder incorporated in cocoa butter being used in finishing. Fine sandpaper disks and cocoa butter are used on labial and lingual surfaces, the operator completing the operation with cuttlefish disks and creating the highest finish possible. Heat from stones or abrasives should be avoided.

INDICATIONS FOR THE USE OF SILICATE

While silicate may be used in pits and fissures, I do not recommend the material for these fillings. I believe that all small fillings, as well as extremely large ones, should be made of metal. Small fillings are best, more nearly permanent and more beautiful when filled with foil. While Class 4 cavities may be repaired with the combination of gold and the inlay surfaced with silicate, it is my belief that, in most of these cases, a baked porcelain jacket crown produces by far the most satisfactory results. I have little regard for a silicate crown as a permanent restoration. Its use is sanctioned only as a temporary measure.

The substitute of the silicate for a broken facing is, in most cases, a makeshift repair, and certainly is not indicated for the repair of modern pontics of a bridge. The material is not adapted as a cement for jacket crowns or inlays. I have successfully used silicate in two surface cavities of bicuspids and molars, but I am not recommending this procedure as the best practice. Here, the inlay is the most applicable. The silicate filling is indicated for Class 5 cavities in some instances, especially if the rubber dam can be applied. These cavities are better filled with foil or a porcelain inlay, and if the cavity covers quite an area, a gold inlay may be inserted. This leaves Class 3 cavities as those for which the most natural indica-

tion is silicate because of its esthetic value and, in many instances, its conservation of enamel. The patient who insists on the elimination of gold because of its appearance will be served with a porcelain inlay or synthetic filling, and I believe that the synthetic filling in the hands of the great majority of dentists will give better satisfaction than the porcelain inlay. Decay does not attack the margins around a synthetic filling as readily as it does the metal filling or the porcelain inlay. At least, this is my observation. Approximal fillings of silicate have injured the enamel of approximating teeth in many instances owing to disintegration of the surface of the filling. In such cases, the contact is also lost.

ADVANTAGES OF SILICATE

Synthetic fillings may be replaced without the loss of much additional tooth structure, and when the patient has been informed that the silicate cement is in most cases not a permanent filling but has some advantages, the first one being esthetics, the patient may be satisfied with the use of this filling for a period of from three to five years, when a new one can be reinserted. In fact, there is nothing in this world that is permanent. The styles change and the people willingly pay the price. It should not be different in dentistry, but it remains to be a case of education. If the dental profession would at all times inform their patients of the truth regarding each operation and prepare them for future work that is necessary in order that they may keep their mouths in a healthful condition, the financial problem of the dentist and his relation to the patient would be largely solved.

DISCUSSION

George E. Roland, Oklahoma City, Okla.: I wish to emphasize the following points: 1. Dr. Rinehart says that in Class 3 cavities we find the most natural indication for silicates. This, I feel, is correct. 2. Class 4 cavities, as Dr. Rinehart says, can be repaired with a combination of gold and the inlay surfaced with silicate. It has been my experience also for this class of cavity that the porcelain jacket crown is much more satisfactory. 3. In my experience, the best results have come from adherence to the following rules: always use the rubber dam; never put silicates in cavities where they are not indicated; have the cavity lining in, the slab and instruments cool, the silicate liquid and powder on the slab, the thin celluloid strip in place, then mix the silicate as thick as possible, leaving just enough time to place it in the cavity and draw the strip up tight around it before the setting starts. I am sure that more silicates are mixed too thin than too thick. After the material has set, I flow paraffn or thick cocoa butter on the filling, and let it set for thirty minutes; then remove any excess material, remove the dam, make sure the occluding tooth does not strike the filling; dismiss the patient and finish the operation at a subsequent appointment. Silicates applied in this way have, in my hands, been very satisfactory.

E. L. Knox, Dallas, Texas: I never advocate the silicate filling. It is almost obsolete in my practice. Its use, I think, should be confined to the proximal cavities in the anterior teeth. Why use synthetic cements when gold foil is so satisfactory?

A. R. McDowell, San Francisco, Calif.: Dr. Rinehart makes no extravagant claims in behalf of this filling material, and, as beyond question, his personal experience is more extensive than that of the average of us, we can safely accept his statement that this material has not yet passed the experimental stage. For purposes of judging the silicates, let us quote Dr. Black's requirements of a filling material, and see how these materials measure up to them. "The qualities most desired in a filling material for *permanent* operation are: indestructibility in the fluids of the mouth, adaptability to the walls of cavities, freedom from shrinkage or expansion after having been made into fillings, resistance to attrition and sustaining power against the force of mastication. . . . The qualities of secondary importance are color, or appearance, non-conductivity of thermal impressions and convenience of manipula-

tion." I leave the verdict to your own judgment without argument for or against as to how well silicate cement meets these requirements. If we are to accept Dr. Black's classification of filling materials, the silicates fall in the class of materials used for temporary operations. I am afraid that the average practitioner does not distinguish between permanent and temporary uses when employing silicate cement in the same sense that Dr. Black does. The silicates have slipped into the dental cabinet and, at the same time, through usage and the manufacturers' claims, into the consciousness of the profession as a permanent filling material, when it should not be so regarded in comparison with foil, amalgam and the inlays. Neither does it measure up to them in fulfilling the requirements of a permanent filling material. J. H. Phillips, of Nashville, stated in a paper read before the American Institute of Dental Teachers at Omaha in 1923, "The manufacturers represent the driving force behind the silicates. They have given to their product names which appeal to the imagination, and persist in claims that practical experience does not support. Through some of the smoothest propaganda ever conceived, these manufacturers have so caught the eye and ear of the profession as to sell tons of their products. A little merit, wonderful claims, and constant pushing, with a solicitous attitude toward good dentistry and public welfare, have done the work." Dr. Rinehart says, "The use of the silicate cement as a filling material has not as yet passed the experimental stage. Nevertheless, it is with us as one of the filling materials in general use." Just how general the use of the silicates is debatable. It is gratifying to note, on examining the requirements of the state boards of dental examiners of the various states, that the majority continue to require gold foil fillings. Only fifteen specify silicate fillings. The dental colleges, too, give gold foil first place over all filling materials, although they teach the use of them all, of course. It seems to me that we may be faddists. Every dentist has his pet ideas regarding his practice. Some use hecolite, others condemn it. Some advocate gold inlays, and others prefer foil. Dental amalgam was bitterly fought by the profession on its introduction, yet it has won second place as a filling material. So perhaps the silicates may come to be looked on with more favor in the years to come. For the present, we must judge its dependability on an analysis of what is now known regarding its qualities. The use of silicates is indicated chiefly because of its esthetic properties, ease of manipulation and insertion and the fact that less care is required for the cavity preparation. All of these are considered qualities of secondary importance in Dr. Black's requirements of a filling material. The contraindications for its use are: It is not a permanent filling; it will discolor; it lacks edge strength; it will disintegrate in the fluids of the mouth; in deep cavities, it endangers the life of the pulp, and, in proximal cavities, it makes a poor contact point. Dr. Rinehart has about limited the use of silicates to Class 3 and Class 5 cavities, and the prime reason given for its use in these locations is its esthetic properties and the ease of replacement in from three to five years. This gives the use of silicates in a nutshell, and undoubtedly will make the advocates of more lasting materials writhe when they recall countless fillings doing excellent service after twenty-five, thirty-five and even forty years. Time brings great changes and dental practice, I presume, will change also. Who can forecast its future? Shall permanent results, so long the objective of our profession, give way to operations of less durability in operative, prosthetic and crown and bridge work, just as is noted in other lines of endeavor?

Dr. Rinehart (closing): I wish further to emphasize the statement that silicate fillings used by the average dentist do not permit a permanent result; this particular point has been emphasized in both discussions. Dr. McDowell spoke of state boards requiring silicate cement fillings for the efficiency test of recent graduates. It is, I feel, unfortunate that any state board requires this operation as a test for ability to practice dentistry. Cavity preparation for the silicate filling and the insertion of the filling do not come within the scope of the fundamentals of scientific dentistry. Therefore, it is my opinion that it should not be included in such requirements. The fact that a graduate is going to use silicate fillings in his practice does not justify a state board test for the operation any more than an examination in the use of the various root canal preparations. A test for a root canal operation would be perfectly proper, but I hardly think that specifying someone's particular formula for the filling requiring a special technic would

be indicated as a test by a state board. There are many practices of advantage to the general practitioner that do not come within the scope of an examination; and a graduate will do many things that his instructors, and no doubt the state board, will disapprove. The teaching of dentistry to undergraduate students includes those principles which have been definitely accepted, and representing the conclusions and findings of our research.

DIMENSIONAL DENTAL CASTING*

By W. S. RICE, D.D.S., Chicago, Ill.

THE subject of casting to dimensions, particularly the casting of single tooth restorations, has, for several years, engaged the active attention of the profession, and it still holds a prominent position among the problems of restorative dentistry for which scientific solutions are being sought. As a result of this widespread general interest and the time and study devoted to the problem by research organizations and prominent members of the profession, standardized procedures are being evolved which no longer limit to a select few the consistent reproduction of wax patterns with dimensionally accurate castings, but place precision casting within the easy grasp of the average operator or technician. Moreover, the success attained is not confined to inlays and other small castings, but has been extended to include large partial and full replacements, which can now be produced as a single piece casting with equal facility and accuracy.

Among the important factors in our present development of a practical scientific casting technic should be mentioned (1) the establishment of sound fundamental principles through the research work of numerous investigators and particularly the work done at the National Bureau of Standards; (2) the growing familiarity of the profession with these principles and their more universal application in adopted individual technics, and (3) improvements in materials and equipment and, perhaps most important, the introduction of mechanically performed operations wherever possible for greater uniformity and precision, in order to eliminate the human variable.

In an earlier paper, I outlined the steps in the casting technic.[1] This outline is repeated here to recall the procedure:

1. Secure the wax pattern by any satisfactory method.
2. Attach the sprue former by adding wax at a convenient point selected for spruing and firmly attaching the sprue to the added wax.
3. Mount pattern on sprue former at position to approximate the center of the investment mass in the ring.
4. Cleanse the pattern with a solution of Gold Dust Twins washing powder or by some other suitable means.
5. Place proper proportions of water and investment in the bowl of mechanical spatulator, incorporate all powder and spatulate 1,200 to 1,400 revolutions per minute for thirty seconds.

*Read before the Section on Partial Denture Prosthesis at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 22, 1930.

1. Rice, W. S.: Casting to Dimensions, Employing a Water Submersion Bath Principle, Dent. Cosmos, 71:960 (Oct.) 1929.

6. From this mix, remove a measured amount of investment to a rubber bowl and add an equal amount of dry investment, by volume, spatulating and vibrating at the same time. This will produce the stiff mix which is employed in investing all cavity surfaces of the pattern and covering all margins. Another way to produce the thick mix is by placing a small quantity of the thin mix in a towel or gauze, expressing the moisture as we used to express the mercury from amalgam. This method of making the thick mix may have the objection of absorbing certain soluble portions of the investment, and thereby modifying its physical properties.

7. Cover cavity surfaces and all margins of the pattern to a depth of one-eighth inch with the stiff mix, vibrating the investment to place.

8. Paint remaining surfaces with thin mix and sprinkle with dry powder to absorb excess moisture in the investment and insure smooth surfaces.

9. Fill ring, close both ends with a clamp and submerge in water bath at 115 F. (46 C.) for twenty or thirty minutes. This temperature may be varied to meet individual requirements for proper casting dimensions.

10. Remove from water bath, detach clamp, sprue former and sprue and burn-out in an oven type eliminator at 1,200-1,400 F. (650-760 C.) for from fifteen to twenty-five minutes, depending upon the type of casting. For a simple occlusal casting, wax elimination will require from fifteen to twenty minutes. Larger castings of the crown type will require twenty to twenty-five minutes. Cast at highest temperature attained in the burning out process.

Under identical conditions, a pattern invested with a single mix of this investment will produce a casting of smaller internal dimensions than one invested with a stiff mix forming the core and covering all cavity surfaces. This is due to the difference in physical properties of a thin and stiff mix and their reactions to chemical and thermal influences.

The stiff mix sets more quickly, is stronger and has a much greater setting and thermal expansion. It would, therefore, offer greater resistance to changes in the wax pattern resulting from structural stresses being released as the temperature is raised for wax expansion. Setting reactions occurring in the stiff mix in advance of similar processes in the thin mix proceed independently of the surrounding investment to form a mold, which is, in effect, the same as if the painting investment had been permitted to set before filling the ring. Changes and distortions resulting from ring resistance to expansion are thus limited to the thin mix.

It has been stated that a stiff mix offers greater resistance to wax expansion and inhibits this compensating reaction, but the fact remains that casting dimensions are dependent on investing temperatures. A pattern invested at proper temperature produces a casting of the required dimensions; whereas, identically the same procedure carried out at room temperature produces a casting of insufficient size.

It has been possible in the research laboratory to execute mechanically practically all operations in the foregoing casting process after the pattern has been mounted on the sprue former. An investment of standard quality and known properties is used, and means are provided for proportioning accurately the powder and water, thus insuring quality and consistency of mix. Mechanical spatulators have standardized the mixing technic, which regulates setting time and gives a plastic, air-free mix. Investing of the pattern is accomplished by a few turns of an investing machine. Expansion of the wax pattern over the required temperature range is accurately controlled by submerging the invested pattern in a water bath of proper temperature. Wax is eliminated in an electric oven which operates automatically at a practically constant temperature. Casting is done in an electrically operated casting machine equipped with a muffle for maintaining

the mold temperature and a muffle for fusing the gold in a favorable atmosphere and at a predetermined casting temperature, the gold being automatically cast into the mold when the casting temperature is reached.

With a wax pattern mechanically reproduced in the research laboratory an accomplished fact, the first and perhaps most important step has been taken toward the development of a practical technic in which the most important operations will be performed mechanically. As a matter of fact, all of the above-described mechanical operations, except investing and casting, have already been reduced automatically to routine practice.

Fundamental principles are well established; standardized materials have been produced and their physical properties published. It now remains only for some individual or the profession to work out a practical technic embodying a mechanical investing and automatic casting process and combine them with selected methods from present procedures to enable a relatively inexperienced assistant to produce results comparable with those of an expert technician. Identical results may be obtained in practical cases as with steel dies or technic models; and, furthermore, any assistant with a little training can attain successful results.

A practical casting technic must possess simplicity, accuracy and consistency; be controllable under conditions of definite precision, and require the least disturbance of the established routine practices. By means of standard preparations on steel dies, the relative value of a casting technic can be accurately gaged by the precision and consistency with which the die can be fitted, following closely mouth temperature and conditions. If a technic does not possess the accuracy and the precision of control to produce consistently castings to fit a steel die, it does not meet recognized present-day standards and should be valued accordingly.

In order to produce a casting having the same dimensions as the wax pattern when removed from the vacuity or die, it is necessary to compensate for the 1.25 per cent casting contraction of gold. This can be attained by utilizing the setting expansion and thermal expansion of the investment and thermal expansion of the wax pattern. The thermal expansion curves of selected investments show that, at a temperature of from 1,000 to 1,200 F. (540-650 C.), a compensating expansion of approximately 0.6 to 0.7 per cent will be realized. The remaining 0.5 to 0.6 per cent necessary to compensate for the casting contraction of gold must be obtained from the setting expansion of the investment during the investing process and expansion of the wax pattern. The setting expansion of investments varies, a value of approximately 0.25 to 0.30 per cent being frequently encountered. The rôle which the setting expansion assumes is not so clear cut as has been previously supposed. A fuller consideration of this phase of the problem is discussed later in this paper.

Published reports[2] show the thermal expansion of wax over the temperature range employed to be approximately 0.03 per cent per degree Fahrenheit. Our experience with the many methods advocated for expansion of the wax pattern has proved that, to date, only one of the methods possesses the necessary accuracy of control, i. e., the water submersion bath, and this is the principle around which our technic is constructed. Expansion of the wax pattern in the investing process is made the elastic link in the chain of procedures, starting with the

2. Research Paper No. 32, Bureau of Standards.

production of the wax pattern and ending in its reproduction in gold, because it can be regulated with precision and may be used to correct any discrepancies that might creep in as a result of individual methods. For the purpose of discussion, I shall divide the casting process into three principal stages: (1) the operations leading up to the production of a satisfactory wax pattern; (2) the investing wax-expansion step, and (3) the burning out and casting step. If a standardized routine procedure is developed for producing wax patterns, wax elimination and casting, employing equipment ordinarily available in the dental office, and these processes are executed in exactly the same way each time, any discrepancies in the compensating factors of a casting technic can be controlled by adjusting the temperature used to expand the wax pattern. The procedure simply resolves itself into the employment of a suitable water bath temperature for a particular individual technic, say 115 F. (46 C.). If, with the technic employed in making the wax pattern and in casting operations, the resulting casting is too small, the temperature of the bath is raised one or more degrees as may be necessary to produce a casting of the desired dimension. If the resulting casting is too large, the temperature of the bath is lowered one or more degrees as required. While special equipment is not essential in other steps of the technic, a water bath with automatic thermostatic control for regulating the temperature is most desirable. For experimental castings, a satisfactory water bath can be made by the use of a container for water and an accurate thermometer, water in the container to be kept at the desired temperature by any means available; but this experimental method could not be considered convenient for routine practice.

The investing temperature is the major controlling factor in any compensating technic because the greatest dimensional changes take place in the wax pattern itself. As stated above, the thermal expansion of wax is approximately 0.03 per cent per degree Fahrenheit, which is almost 200 times the thermal change which takes place in investments for the same temperature range. While the expansion of wax is such an important factor, it is at the same time the easiest to control. It has been pointed out[3] that "the field of operation is relatively constant at 95 F. (35 C.) and it is from wax impressions at this temperature that compensation calculation must be made." It is further stated "no gold inlay technic can possibly reach the maximum efficiency unless zero dimensions, i. e., the point from which dimensional changes plus or minus are calculated, is placed at tooth cavity temperature which, in the bicuspid and molar region, is, as previously stated, approximately 95 F. (35 C.). Hence, the only wax movement which can be utilized in neutralizing the casting shrinkage of gold is the expansion which occurs at temperatures above 95 F. (35 C.).

Since the temperature at which a pattern can be more easily secured seems well established at 95 F. (35 C.), this temperature has been accepted and is used as a basis for the compensation calculations here presented. No great difficulty should be experienced in adjusting the individual wax pattern technic to the standard one of removing each pattern from the cavity at this temperature. As a matter of fact, we are led to believe that it is not at all easy to obtain patterns below this temperature and that to attempt to do so only adds to the difficulty of the

3. Van Horn, C. S.: Dent. Cosmos, 17: 764 (July).

procedure and introduces another variable and possible cause for failure. The point to be emphasized is that the procedure of producing a wax pattern in the mouth can be readily limited to this temperature with the least modification of the established routine.

By expanding the wax pattern over a predetermined temperature range from the temperature, i.e., 95 F. (35 C.), at which it is removed from the cavity, a definite amount of expansion is accomplished each time which is transmitted to the resulting casting, but the changes which take place in the wax with relatively small changes in temperature are so great that accurate control of the expanding temperature is essential. Many methods of procedure have been suggested and carefully tried out in our laboratory, but the desired temperature could be obtained and consistently controlled only by using the water bath principle.

Some of these methods were discussed at length in a previous paper,[1] and the conclusion was reached that hot air ovens are hopeless. Likewise, the expectation of starting with an investing temperature sufficiently high to reach 115 F. (46 C.), exactly at the same instant the investment begins to set, seems equally impractical.

From the foregoing, it will be noted that to produce castings of predetermined dimensions, the operator simply follows his usual routine of producing a wax pattern, burning out and casting. These operations cannot be performed in a slipshod manner, but must be reduced to a standard procedure identical in each case. The precision of the results will naturally depend on the care and precision with which these steps are executed.

In the investing process, there should be adopted and used a standard investment which is uniform in quality. Also, a water bath temperature which gives the desired results for the technic employed, should be determined by practical experimentation.

It has been suggested by some authorities that our difficulties with the casting problem would be forever solved if an investment could be secured with a thermal expansion which could be utilized in the casting process, and which is equal to the casting contraction of gold. Recent investigations at the Bureau of Standards have disclosed not only that it is possible to secure an investment with this amount of thermal expansion, but also that they have actually produced an investment which has a thermal expansion of 1.4 per cent when heated to approximately 300 F. (150 C.). Still the casting problem has not been solved. A wax pattern secured at 95 F. (35 C.) would have to be invested at exactly the same temperature to utilize fully the 1.4 per cent thermal expansion of the investment and still produce a casting of proper dimensions. If, for instance, the pattern was permitted to cool to room temperature and invested at this temperature, say, 75 F. (24 C.), a contraction of the pattern over the temperature range from 95 F. (35 C.) to 75 F. (24 C.) or 20 F. (11 C.) would result.

This would amount to a contraction of 0.6 per cent, which, added to the casting contraction of gold, would give a total contraction of 1.85 per cent. An investment with a thermal expansion of 1.25 per cent, or even 1.40 per cent, would fall short of accurate compensation by 0.40 to 0.65 per cent.

SPRUING

Several methods of attaching sprues to the wax pattern have been tried, including attaching the sprue to wax added to the pattern for the purpose, and also driving the sprue into the body of the

pattern. Indications are that comparatively little, if any, distortion occurs by spruing to added wax; whereas, driving the sprue into the pattern invariably produces distortion of greater or less degree. In the investigation of this problem, a pattern of the mesioclusodistal type with straight approximal arms and occlusal surface formed over a square die was used. The pattern was approximately three-fourths inch in all outside diameters by five-thirty-seconds inch in thickness. It was formed by pouring melted inlay wax into a sleeve surrounding the die and tempered in a water bath at 95 F. (35 C.) before removal from the die. In shape, the pattern is most favorable for changes incident to spruing, but, in the method of forming the pattern and in dimensions, it is probably more resistant to the effects of spruing than patterns formed in practice under pressure and varying in thickness in different portions.

All measurements in the determination were made at 95 F. (35 C.), the pattern being carefully measured before and after attaching the sprue. Sprues attached to an added quantity of wax proportional to the size of the sprue and pattern showed slight changes in the total mesiodistal outside and inside dimensions of the pattern at the gingival aspect. Patterns to which the sprues were attached by imbedding the end of the sprue in the body of the pattern about the center of the occlusal portion invariably showed an increase in the outside dimensions of the approximal arm, and a proportional increase in the distance between the arms, but approximately no change was noted at the occlusal surface. This would indicate that mesioclusodistal, mesioclusal or distoclusal patterns may be expected to distort varying amounts if sprued directly into the wax. This distortion ranges from 0.10 to 0.5 per cent, depending on the variables of temperature and size of sprue, dimensions of pattern and other conditions. The amount of distortion is conclusively shown to be greatly reduced by attaching the sprue to wax added at the point of spruing.

INVESTMENT OF THE WAX PATTERN

Just what happens to the dimensions of a pattern when it is invested has never been satisfactorily determined. The setting expansion of investments has been measured with the investment unconfined and the assumption has been that the benefits of this expansion are obtained in the investing process. This is the change which is to be expected in an invested mass free to expand in all directions.

In a practical case, the invested mass is confined in a rigid, practically nonexpanding brass ring. In this case, what is the effect of the setting expansion? Is there no expansion, or does the expansion press inward on the wax pattern? In order to test this qualitatively, a tapered brass plug was invested inside a brass ring. When the investment had reached its initial set, the plug was removed, and the investment allowed to come to its final set. After this state was reached, with the nonexpanding ring, the hole was smaller than the plug, and the plug would fit only part way into it. The experiment was repeated with a slotted ring capable of free expansion. In this case, the plug, when placed in position, extended through the bottom of the investment. Thus, it was demonstrated that, in the case of a nonexpanding brass ring, the expansion of the mass of investment took place in such a way as to constrict the hole in the center of the investment.

While the expansion may tend inward toward the center of a hole, the question of the effect of the setting expansion of

the investment on the dimensions of a wax pattern when invested in the usual type of ring still remained to be settled. For this work, in order to measure the dimensional differences, a mammoth crown pattern was made by casting the wax over a large crown die. This wax pattern was then turned on the lathe to give a pattern of more uniform dimensions. After the crown was measured accurately at a given temperature, 77 F. (25 C.), it was invested within an iron ring, a thermocouple being imbedded in the core of the investment about one-fourth inch from the wax pattern, and another thermocouple at the same distance from the outer surface of the crown, the temperature rise on setting being noted. The investment was then carefully cut from the top portion of the ring, the surface of the wax being exposed. Measurements were made of metallic measuring points with which the pattern had been provided, and it was found that with a thin mix of investment, the diameter of the core and the outer diameter of the mold at the gingival margins were practically unchanged. On the other hand, with a thick mix of investment in the core of the crowns and a thin mix outside the crown, an expansion of approximately 0.1 per cent was noted in the core at the gingival margin, but practically no change was observed in the outer diameter. Measurements midway between the gingival and occlusal surfaces showed more striking evidence of the effect of the setting expansion of the investment. Here, with both the thin and thick mixes, an expansion of 0.1 per cent was observed in the core, while a contraction of the same order was found in the outer diameters.

With an expansion of the diameter of the core of the crown and a contraction of its outer diameter, it follows that the wall thickness of the crown has decreased. The decrease of the thickness at the top of the crown is negligible where the thin mix is used, but is approximately 1 per cent where the thick mix is used in the core, and midway between the gingival and occlusal surfaces, the decrease in wall thickness amounts to 2 to 2.5 per cent for the thin and thick mixes, respectively.

Assuming that the actual volume of the wax cannot be reduced under the relatively low pressure here dealt with, it is necessary then to account for the apparent loss of volume of material. Measurements made on the occlusogingival dimensions of the crown show a substantial increase (0.6 per cent) in height as a result of the investing process.

The investing process as it is frequently carried out in a nonexpandnig ring with a uniform investing mass is shown to result in a compression and elongation of the walls of the wax pattern, with an appreciable bulging at the center of the core in the case when the thin mix is used. This bulge appears to be negligible with the thick mix.

It is thus demonstrated that, whereas with the use of a thin mix throughout, the benefits of the setting expansion at the gingival margin are lost and the crown is slightly larger midway between the gingival and occlusal surface than at the gingival surface, the practice of using a thick mix in the core and at the margins of a crown pattern and a thin mix outside affords the benefits of part of the setting expansion of the investment.

THERMAL EXPANSION OF INVESTMENT

One of the chief controlling factors in compensating for the casting contraction of gold is the thermal expansion of investment. The amount of this expansion is governed by the loss of heat after removal of the mold from the wax elim-

inator or the temperature of the mold at the instant of making the casting. Since temperature plays such an important part in precision casting, the heating and cooling rate of a standard size inlay mold and the relation between oven temperature and the temperature at different points in the mold were determined. Temperatures were recorded by thermocouples located between the ring and muffle wall, in the investment near the wax pattern and an outer couple near the inner surface of the ring. The molds were heated in two types of electric ovens, one just large enough to accommodate a single ring, and the other about 4 by 5 inches inside dimension. Oven temperatures were maintained practically constant at from 1,350 to 1,400 F. (730 to 760 C.). The invested patterns were transferred directly from the water bath to the ovens, and temperatures at the two positions in the investments noted at one minute intervals.

It was found that the mold temperature increased more rapidly in the smaller muffle, the center couple indicating 1,000 F. (500 C.) in sixteen minutes. The larger muffle required twenty-nine minutes to raise the center of the mold to this temperature. The average cooling rate of the mold for the first five minutes after removal from the muffle was about 55 F. (30 C.) per minute in the center, and 65 F. (40 C.) per minute near the ring. It must be recognized that these values will vary considerably with different kinds of equipment, and for that reason can be accepted only as an indication of heating and cooling rates. The cooling rate will be further affected by melting the gold in various methods of casting.

THE IMPACTED TOOTH*

By T. A. HARDGROVE, D.D.S., Fond du Lac, Wis.

THE impacted tooth and irregular teeth are, to a large extent, an endocrine problem and not one of resistance of an anatomic nature as many of us are inclined to think. We are inclined to regard the problem as one of anatomic resistance because we must remove tissue in order to remove impacted teeth. We are doing the only thing that can be done, as a rule, when the patient comes to us for relief; and while we have made progress in surgical removal technic, the fact remains that it is still an unpleasant experience for the patient.

Anatomic resistance or obstruction will not explain why teeth occur out of position; nor does it explain why they are misdirected or not directed at all in their effort to take their proper position in the arch. An impacted tooth is one growing without control, and sometimes the force necessary to put it in position is absent.

It is usually the cuspid or third molar that is impacted, for the reason that they are the last to be driven into the arch; and about that time, there is, as a rule, a readjustment of the endocrine activity,

* Read before the Section on Histology, Physiology, Pathology, Bacteriology and Chemistry (Research) at the Seventy-Second Annual Session of the American Dental Association, Denver, Colo., July 23, 1930.

because, at that age, the thymus is apt to be disappearing and its function ceasing.

The first essential in the matter of diagnosis is a consideration of the history of the impacted tooth or teeth, since we find impacted teeth present in different members of the same family. It has been my observation that all the members of the family are liable to have impacted teeth, which might be expected, since the impaction occurs as the result of endocrine inefficiency and imbalance, and endocrine systems and the functions peculiar to that system are inherited.

Let me quote from Kimber and Gray:

> In addition to reflexes our original equipment includes a large group of more complex activities known as instincts. Edward L. Thorndike defines as instincts connections or tendencies to connections which are unlearned —are in us apart from training or experience.
>
> The characteristics of instincts are: (1) they function adequately the first time the proper stimulus is received; (2) they are common to the human race, indeed many of them are common to the higher animals as well; (3) in comparison with the reflexes they are complex, consisting of a chain of movements; (4) they are accompanied by consciousness though not dependent on consciousness for control; (5) they are modifiable.

I am not presenting this quotation to you as an exact scientific explanation of the mechanism of which I am speaking, because it is not entirely that, but rather to stimulate your thought in the direction of an understanding of this problem.

The thyroid is the pacemaker of the endocrines and influences the other glands, probably more than any other. The thymus receives little attention because we have always supposed that it disappeared shortly after birth. We know now that it does not disappear until the age of 16, and, in some individuals, it lingers longer, sometimes all through life. These two seem to have the supervision of the placement of the teeth in the arch. It is said by Reese,[1] of the University of Wisconsin, that the administration of the extract of these glands will cause sluggish or delayed eruption to change so that the teeth take on a renewed activity. In the light of this experience, it would seem that the function of these glands was interrupted or arrested; in the case of the thymus, sometimes permanently and at other times only periodically or spasmodically.

As a rule the tooth without control has lost that influence permanently, but, in some patients, it is renewed spasmodically, which accounts for the so-called pain of a reflex character, as we say sometimes, and because patients have it at certain times and do not have it at others, we say that the patient is neurotic, and when he is relieved following operation, we say that we relieved the mind of the patient. In reality, we have relieved the patient because there is no tooth to drive into position. The endocrine influence which is responsible for the intermittent action of that tooth in its effort to get into position has no more work to do for that tooth.

As the histology is quite simple, I shall not deal with it at length. We are all familiar with the development of the enamel organ from the epiblastic layer. We know that the enamel organ is the pacemaker of the tooth unit, that the other tissues of the tooth unit are developed largely from the mesioblastic or middle layer. If the cells are not directed by a normal inherited action or if there is periodical interference with endocrine function, in particular the function which places the tooth in its proper position, we are apt to have the enamel development

1. Reese, H. H.: Significance of Endocrine and Vitamin Deficiencies as Etiologic Factors in Dental Abnormalities, J. A. D. A., 17:2198 (Dec.) 1930.

without proper direction or control. It may result in the crown of the tooth being placed upside down, so to speak. If, then, there is no other influence that can correct the position of that tooth, the structures surrounding it will cause it to be maintained in that position, playing a part at that particular time, only because the endocrine function is not normal.

If there is resistance or obstruction of an anatomic or mechanical nature, it must be dealt with accordingly. Impactions do not occur in the temporary teeth for the reason that, at that age, the endocrine forces are at their greatest activity if the child is normal and is fed normally. And that brings up the question of diet and hormones, which work in conjunction with the endocrines or as a result of their activity. The direction for the placement of teeth in the arch is determined at conception and is continued to birth, and it is so arranged that the mother will nurse the young for a time, and her milk contains not only the elements necessary to produce minerals and other matter, but also the hormones and vitamins, so essential for proper jaws and teeth and proper placement of the teeth in the jaws. Breast feeding should be carried on until the child's endocrine function is established. The child itself is the best judge of that time. Dr. Barrett of Buffalo used to say that a child should be fed at the breast until it refuses it. The English and American people have poor teeth because the nursing period is short and in Latin and other countries, better teeth and fewer impactions and irregularities are seen, because breast feeding is continued over a much longer period. Without a sufficient period of breast feeding, each generation will have more irregularities, and more impacted and decayed teeth. The American mother must save the teeth of the race by longer nursing periods, so that a better endocrine system and better functioning of that system may be established. Decay, irregularities, impactions and deformity of the jaws are, from the endocrine point of view, a related problem and should be dealt with from that angle. We can to a larger extent than we suppose practice prevention.

PATHOLOGY

A great many conditions are attributed to the presence of the impacted tooth, many times wrongfully and sometimes correctly. If the impacted tooth is entirely surrounded by bone with no possibility for infection to enter from the outside, and if it does not cause pressure on neural tissue, it is not apt to act as a source of focal infection or irritation. The history of these cases shows that people go through life with these teeth causing no disturbance. If the tooth is braced against the tooth in front of it and the endocrine system is still active, while the root of the tooth is developing and for some time thereafter, there may be spasmodic and irregular efforts to drive the tooth into position, which will account for the reflex pain present. And with those symptoms, the impacted tooth is an expression of the fact that there is still an effort on the part of the endocrine system to perform its function and, in some individuals, that function is carried to the point of an actual pathologic process in the endocrines themselves, due to an overproduction of the chemicals necessary for that action. That is the principal involved when, in the eruption of teeth, children have convulsions. The convulsion is due to an overactivity of some endocrine gland creating an imbalance. The resistance offered in the eruption of the teeth calls out a hormone which is responsible for that action. As quickly as the resistance is removed, the

convulsion stops. According to Reese's statement, an excess of one vitamin can suppress the activity of another, and that may hold true in the case of the hormones. The hormones, which are simply a substance manufactured in one gland or organ, will pass through the circulation to another organ or gland and produce an action in that organ.

That is demonstrated when Nature makes a continued and extreme effort to push the tooth into position. There is, of course, no denying the fact that if it is possible for the infection to find its way from the mouth down to the crown of the impacted tooth, it can surround it and act as a source of general infection, and then we are dealing with a focal infection just as in a tooth with an abscess or granuloma. Sometimes, an impacted molar will bring pressure on the inferior or mandibular neural tissue, and a persistent pressure on that tissue will cause local pain, and may eventually produce some changes in the cells receiving that irritation in the brain itself. Dr. Cotton has reported many cases of insanity caused by that type of disturbance. If those patients were cured when teeth were removed, it would indicate that the dental condition was the cause of the insanity. We might argue also that the impacted tooth is but an indication of an abnormal histologic and embryologic action and that the insanity occurs as a result of a continued imbalance of the endocrines. When we come to study the impacted tooth, we find that it furnishes to the dental profession a field for investigation that is beyond any other problem.

Leaving that view of the subject, what can we do for the patient who has or is going to have impacted teeth?

The first thing is diagnosis. That should be done at an early age, as soon as the child has begun artificial feeding. Roentgenograms should be made at intervals and kept as a record. The important thing, if we are thinking of surgical relief for the patient, is to discover the condition before the roots have formed so that the tooth will not be so hard to remove.

If we wish to influence the early endocrine chemistry of the body, or I might better say, the endocrine chemistry of the mother before the child is born, it should be done at conception and when child bearing is anticipated. That is the time to make an effort in the direction of endocrine control through the medium of health and a diet containing vitamins and by the use of endocrine therapy, in the hope that it will influence the hormones. Tooth buds are laid down at a very early period and they are apt to follow the direction of the early endocrine dictation of the mother and father, and so the problem becomes a real one. As endocrine therapy is not entirely practical yet, we must still rely on surgery for the relief of most of these conditions. By that, I mean that the time to remove impacted teeth, particularly the molars, is as soon as it is practical and at a period when it is safest for the individual, and before the roots are developed. That period varies in different individuals. In some, it may occur at 14, 15 or 16 years of age, or up to 20, and more often at 20 than we are inclined to think.

It is a wise plan to wait until after the age of 16 or at least until we have reason to believe that the activity of the thymus gland has ceased to be a factor, and at such time that we can have the cooperation of the patient if we are using a local anesthetic. The average patient will go along safely until 18 or 19 years of age before there is a root developed on an impacted third molar. At that time, it is quite a simple matter to open the tissue

in that location and remove the crown and sack in which the tooth is developing, the further development of that tooth, which is responsible for the disagreeable features associated with the removal of it when the patient is older, being thus prevented. It is the root of the impacted tooth that makes removal difficult. In many cases, it is an operation associated with more possibility of trouble than we are inclined to believe, the contention being that these teeth should be removed before there is a root developed on them, and thus prevention be practiced to the extent that we get away from surgery necessary in the hard dense bone laid down when the root is so fully developed, and also from the pathologic process that is likely to occur if it is allowed to remain. Impacted cuspids as a rule do not cause a disturbance unless the roots of the bicuspid or other teeth impinge on them, and through wear and usage, they eventually become a source of irritation; or if the other teeth have been removed, they are apt to act as an obstruction to artificial dentures. It is a wise policy in the treatment of impacted teeth and particularly cuspids and central incisors, if they are not making a disturbance and the patient can get along comfortably with them, to allow them to remain. The proof of this is exemplified in the fact that so many people never know they have impacted teeth and particularly impacted cuspids.

It is much easier to remove just the crown or the enamel than to deal with the tooth after its roots are long and malformed and a harder bony structure will obstruct its removal.

Malformation and irregularity of roots are caused by a lack of, or poor control of, the endocrines. The impacted tooth requires daily attention, and yet we have done nothing to prevent the hard or disagreeable features of its treatment. We have made no more progress than the physician has in the treatment of appendicitis. We have had for our care a condition that we could, if we made the most of our possibilities, prevent, at least in its worst features. I mean to say that when a tooth is found to be growing crooked, we should know it and at once do something to relieve the situation. At any rate, we should know the family history and make an effort to correct endocrine activity for the members of the family yet unborn.

Nature has, in her wise scheme of creation, left it possible for us to prevent a deep impaction with long roots surrounded by hard bone. To begin with, the first thing that is developed within the sack is the enamel and that is closer to the surface at from 15 to 20 years than it is later. In the case of an impacted tooth, the roots grow down deep into the bone and the enamel cannot erupt because it is held back by something pressing and fixing it in one position, owing to the fact that it was started crooked. The enamel does not change, but the root does. It is the only thing that can. It is quite a simple thing at that early age to remove because we have only to remove the crown.

Teeth can be impacted without being malposed. Nor need they be misdirected: they can remain straight, if the force of resistance is equal in all directions. They may be without the force that should drive them up into position.

Take, for instance, for an endocrine example on a larger and somewhat different scale, the case of a litter of young pigs. There is always one and many times more than one that is what is commonly known as a "runt." That is an expression of the principle that the mother of the young pigs has exerted all of the endocrine influence that it was possible for her

to give up at that time, and it is a principle in nature to overproduce. The one pig is a so-called "runt" because it has not received its proper share of endocrine assistance.

The prevention of an impaction appears to be one of control of the endocrine gland activity and particularly of the thymus and thyroid, with emphasis on the thymus. By the proper use of the extracts of those glands, we will eventually enable patients to grow bones and well-developed and properly directed teeth and thereby prevent the impaction of the tooth by assuring it a normal position. While it is true that we do find apparently normal jaws, as far as appearance is concerned, in which are impacted and imbedded teeth, this fact does not dispute the principle laid down that the same influence that produced the apparently normal jaws should have placed the teeth in their normal position.

I should like to say again that the cuspid and the third molar are the teeth that are impacted as a rule, because there is a cessation or hesitation in the functional activity of those glands; or, if you like, a disturbance in the intrabalance of all the endocrine glands, which, of course, could be due to many things. It could be due, for instance, to some of the childhood diseases, and it could be due to some of the chronic inherited diseases, such as syphilis. Its presence does not imply that the individual must have any of those diseases in order to have an impacted tooth because, in the normal individual, owing to diet and vitamin influence and hormone activities, which are interrelated, there may be a hesitation in the endocrine process that would result in an impacted tooth.

The enamel is the pacemaker of the tooth unit just as the thyroid is the pacemaker of the endocrine glands. While all of these tooth buds are laid down early and nearly at the same time, the impaction remains to be developed in the second set of teeth, owing to the fact that the child is apt to have a more nearly normal function of the endocrine system develop up to 2 or 3 years of age. I mean that Nature makes her greatest effort to harmonize and develop the endocrine system up to that age, and, in a normal individual, that endocrine activity continues until the age of 16, i. e., until the thymus ceases to be active, a critical period in adolescence.

Orthodontists know that if they stimulate crooked teeth in certain individuals they will virtually take their proper place in the arch. They are awakening a dormant endocrine in that patient by causing a hormone to be produced that demands the action of that force. They call it stimulating tissue. When orthodontia comes into its own, it will do wonderful things. As it is at present, it is on a par with other dental repair work. Mechanics has its limitations; the other side of dentistry, the so-called scientific side or research, has an unlimited field, and progress from now on must be made by it.

CONCLUSIONS

1. Impaction occurs as a result of endocrine imbalance, inefficiency and intermittent action.

2. Anatomic resistance is only secondary and would not be a cause if the endocrines functioned properly.

3. An impacted tooth is an organ developing without control or without direction and sometimes developing without the force generally supplied to teeth for their proper eruption and placement.

4. The force and control is intermittent in some individuals and accompanied by spasmodic pain.

5. Endocrine therapy, diet and hormone stimulation may be of assistance if

given early, and will, when we understand it, prevent impactions.

6. Impactions are likely to be the result of an inherited influence and an early check-up should be made.

7. It is a mistake to allow the root to develop when the tooth is sure to be impacted.

8. It is easier to remove the crown only than it is to deal with it later on when bone is hard and roots are deep and impacted in the bone. The operation should be done when only the crown needs to be removed.

9. A great many impacted teeth can be removed as late as at from 18 to 20 years of age, before the roots have developed. The roentgenogram will determine that.

10. The thymus and thyroid are the endocrines and the important glands in the impacted problem.

11. The cuspid and third molar are likely to be impacted because they are the last to take their proper place, at about a time when the thymus is becoming obsolete and its influence is not marked.

12. Repair and correction of irregularities of teeth and jaws, and even surgery, are only reparative and not basic in the effort for the correction of dental ills.

13. About the time the thymus disappears and its function ceases, the teeth should all be in position.

14. Eventually, a proper extract of the thymus and thyroid, particularly the thymus, will be produced that can be administered to patients and will prevent the impaction of teeth.

DISCUSSION

Donald M. Gallie, Jr., Chicago, Ill.: There is perhaps no factor in human existence, in racial development, in the advancement of civilization or in the mental and physical development of the individual more fundamental than the interrelation of the secretions of the ductless glands. To what extent the functional activities of these organs have influenced the development, change and stability of the animal world cannot at the present time, or by our present knowledge, be ascertained. The science of endocrinology has opened up fields of speculative thought and practical application the possibilities of which are staggering to the imagination and conception of biologic control. When we realize that the rational study and observations of these most complex structures are the latest development in the world of medical and scientific thought, it is but natural that we should be impressed by the enormity of our ignorance regarding the function and interrelation of the secretions of these organs. The most learned scientists who are laboring in this field of research, recognize, perhaps more than others, the fact that the primary and most superficial knowledge is all that we have in our possession at this time. Science is progressing at so rapid a pace that while the study of this subject is still in its embryonic state, many contributions have been made which are of distinct value from the standpoint of practical application. The influence of the ductless gland secretions on mental and physical development can be most easily recognized by our observation of the abnormal. Our understanding of the physiologic is greatly augmented by our observation of the results of pathologic conditions in these organs. Multitudinous variations of mental and physical deformity can be recognized as results of endocrine unbalance, ductless gland hypoactivity and hyperactivity. In the more apparent deformities, we have been able, by our present knowledge, to recognize certain factors of definite cause and result, and endocrine therapy has been successfully applied to a vast number of pathologic conditions in the past twenty years. Atypical and asymmetric growth and development is a most common result of endocrine unbalance, and there appears to be a marked condition of definite anatomic selectivity of organs involved in a given type of endocrine disease. It is noteworthy that factors of endocrine unbalance causing a variety of mental and physical deformities so often are associated with atypical, asymmetric and abnormal development of the maxilla and mandible and, with these deformities, a marked tendency for dental malformation, malocclusion and variation

from the normal. In our observations among the insane, in whom ductless gland disease is so often a cause or result, dental and oral malformation can be noted as a most common occurrence. Malocclusion and impacted and supernumerary teeth are present in these individuals out of all proportion to the number examined. Anatomic and histologic deviations from the normal are often observed in the maxillary and mandibular bone structures of these patients as well. The influence of the activity of the thymus gland on dentition and dental occlusion has been clearly demonstrated by Dr. Hardgrove, and its relationship with noneruption and impaction of teeth is clearly understandable in the light of his observations. By the same token, study of dysfunction or abnormality in the pituitary body discloses some interesting facts in mandibular and maxillary bone deformity. Atypical and asymmetric growth and underdevelopment, failure of closure and the facial clefts, associated disproportion of subperiosteal and cancellous bone, are but a few of the oral manifestations of activity of a portion of the pituitary body. Deviations from the normal in maxillary and mandibular bone development will, of necessity, be a factor in dental occlusion, eruption, noneruption, impaction and abnormality in tooth relationship. In reviewing a series of dermatologic case histories in which skin, hair and nail abnormalities had been studied from the standpoint of thyroid, thymus, pituitary and suprarenal unbalance, it is interesting to note that abnormal size and atypical enamel development in the teeth have been recorded by the endocrinologists. We are entering into a new era of scientific biologic understanding. The dental surgeon of our present generation will, of necessity, soon be delving into channels of thought, and working with dynamic forces of biologic activity such as are overwhelming to our present understanding. New avenues of activity are constantly being opened, avenues inconceivable in our present idea as to what constitutes the practice of dental surgery. Statistical records, compiled case histories, research in the fields of medicine, dental surgery, biology, ethnology, paleodontology and comparative anatomy must of necessity be familiar to the dental surgeon of perhaps today and unquestionably tomorrow. It is conceivable that the present presentation given by Dr. Hardgrove may stimulate successful research along the lines of endocrine control, which will make it possible for us to direct the factors of dentition and eliminate many anatomic and functional abnormalities which we are now treating by therapeutic and surgical measures. Dr. Hardgrove has emphasized in his presentation the potentialities of the impacted tooth as a source of neurologic irritation and a factor in local and generalized infection. He also outlined the dangers and possibilities of both acute and chronic bacterial exacerbations centered about this oral abnormality. There are but one or two items I should like to emphasize in this discussion which I do not believe Dr. Hardgrove has found time in his paper to consider. The first is the possibility of cystic development caused by the epithelial elements surrounding the enamel organ and the abnormal stimulation of these epithelial elements by the activities of growth in impaction. Whether the impacted tooth is subject to oral infection or not, I believe this possibility should at all times be considered. Secondly, the almost constant irritation of the mucoperiosteum encompassing impacted teeth, with its usual condition of hyperplasia, and epithelial derangement, is highly conducive to neoplastic change, cell and tissue degeneration. I am in accord with the idea that surgical removal of impacted teeth is clearly indicated at an age when the roots have not fully developed, which eliminates a most important mechanical difficulty in the successful handling of the case. There are many other considerations which would indicate that greater success may be anticipated when the condition is corrected during the period of youth. The resistance of the young individual to surgical shock, his greater adaptability to both general and intraneural anesthesia as well as his resistance to both local and general infection are factors that make him a preferred surgical risk as compared to one in more advanced life. Because of the predominance of the organic components of the inverting bone surrounding impacted teeth, less resistance is met with in necessary osseous resection. By the same token, more speedy, physiologic bone repair can be expected as a postoperative result. Because of the greater proportion of vital cellular elements in the bone of the young individual, fewer osseous complications are encountered and a better prognosis may be expected than in the older subject. Another rather valid reason for our advocacy of the removal of impacted teeth before the

complete formation of the tooth roots is noted particularly in the impacted third molar. The relationship between the fully developed roots and the inferior dental canal with its large nerve trunk, blood vessels and lymph accessories is far more intimate than during the early period of tooth development. The injury of these structures during and after third molar enucleation is most distressing to the patient, often resulting in many varying ramifications of neurologic discomfort over long periods of time. I am in accord with Dr. Hardgrove's belief that endocrine activity is perhaps the most potential factor in abnormality of dentition. I believe there are ductless gland activities which are accountable for deviations from normal in the anatomy, morphology and structural histologic elements in the tooth *per se*, as well as other quite different endocrine activities that determine the facial, maxillary and mandibular bone development. In the study of ethnology, we are conversant with certain normal racial characteristics and deformities which are constant in a given type of man. In our researches in paleodontology, certain peculiar normal characteristics are noted, and certain pathologic deformities predominate at different periods in the development of the human type. Deviation from the accepted normal are noted in the structure, size and characteristics of the human teeth themselves. Variations in the hard palates, mandible and mental protuberance constitute recial and paleodontologic facial characteristics. To go a step farther in our understanding of ontogenetic and phylogenetic correlation in the development of the human being, it is easily understandable why we often encounter end organs, and anatomic deformities traceable to our progress through the intricacies of change of species and development from primitive man and the lower animals. That endocrine and hormone activity is the predominating factor in the development of the human species, and is accountable for certain reversions and stigmas of degeneraton found in the unfit and undeveloped, seems a reasonable hypothesis. Much additional information and knowledge must be compiled before our understanding of these great fundamentals of biology can be definite. The incidence of abnormality of dentition and impaction in geologic relationship must be compiled and statistics recorded. The incidence of impaction and dental anatomic deformity in existent races must be understood. The effect of different environmental and nutritional factors must be taken into consideration and associated with all the endocrine findings before the entire story is disclosed.

NUTRITIONAL FACTS THE DENTIST SHOULD KNOW*

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TREMENDOUS changes have taken place in the world within the past twenty years. The radio, motor car, airplane and other inventions have revolutionized modern society. A new generation is in the saddle. What does this new generation expect of dentistry? To attempt to meet the new conditions in the old way would only result in failure.

What does the patient 30 years of age, who twenty years ago was taught oral hygiene in public school and was required to come in for periodical examination of his teeth during school hours, expect of his dentist today? What are the cumulative effects of years of public instruction in oral hygiene through the press, the air, the platform and dental clinics? The dentist is meeting a more enlightened public, one that has been taught oral hygiene, is endeavoring to practice it and

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expects the dentist to play his part in the preventive program.

Human nature is so constituted that new ideas or methods that appeal to the popular fancy are played up and exploited in such a way that their importance is magnified beyond all reasonable limits. Scientific and professional men are only human, and many of them have become notorious as hobby-riders. This may be the way of progress, but one would prefer a more orderly advance. The hobbies that have been ridden by dentists are legion. There are hobby-riders in preventive dentistry as well as in restorative dentistry, and we have also had our hobby-riders in bacteriology and pathology and in nutrition.

From the time of Lister, and particularly during the past twenty years, the pendulum swung far in the direction of a bacteriologic concept of disease. Bacteria have been painted as arch enemies of mankind, with no useful place in the scheme of things. This is not only unscientific but also quite impossible. Our earlier ideas regarding bacteriology led us to regard the body as a walled fortress, surrounded by a host of attacking bacteria, the very life of the city depending on whether the invading army was repelled or not. Focal infection was merely a shifting of this emphasis from a massed attack of the enemy to unheralded and surreptitious entry at a localized point, somewhat akin to spies' penetrating the defenses and then causing serious trouble within the beleaguered city. It is not my desire to minimize the place of bacteria as a causative factor in disease, but rather to caution against the extreme view that attaches all importance to considerations of bacteriology and little to physiology.

May we not properly inquire as to whether the "beleaguered city" has not been overly enthusiastic in its attempt to utterly crush the enemy? Bacteria cover the skin, are present by the tens of thousands in the mouth and lungs and are found throughout the alimentary tract, where they appear to have some useful function. They are even found at times in the blood itself. Nature has built up protective agencies in order that the body may cope with bacteria.

When disease ensues, we must ask ourselves: "Is this due to the force of the attack or to the weakness of the defense?" While we must continue to consider the "enemy at the gate," should we not give more study to disintegrating forces within, forces that lower the resistance? Furthermore, we have come to learn that many diseased conditions which were formerly thought to be caused by bacterial toxins are caused by chemical toxins of metabolic origin within the body. The pathology of the future, then, must distinguish between conditions that relate, on the one hand, to bacteriology, and on the other hand, to metabolic toxins.

A committee of American and Canadian physicians, in cooperation with a similar committee in Europe, has been working intensively on the cause and treatment of arthritis. A great deal of progress has been made, and the fact has been established that arthritic conditions are not always caused by bacterial toxins but are frequently due to chemical toxins, induced by wrong habits of living and particularly by an unbalanced diet.

Ralph Pemberton, in the November issue of *The American Journal of the Medical Sciences,* has shown not only that chemical toxins may be the causative factor in bodily disturbances (which may, in turn, result in bacterial invasion), but also that the chemical toxins are the cause of many disease conditions.

Heretofore, arthritis, which is more prevalent than tuberculosis, was con-

sidered to be due entirely to infection. In the search for localized infection in the body, the roentgenogram has centered special attention on the teeth, many of which have been sacrificed without direct evidence of their involvement.

The newer work, which shows that arthritis may be due to chemical toxins not of bacterial origin, may possibly explain why the extraction even of infected teeth may result in no improvement in the condition of the patient.

To quote from a report prepared by the American Committee for the Control of Rheumatism: "The commonest causes of arthritis are the poisons which are set free in our bodies. These poisons may be manufactured by the growth of germs which the body's defense mechanisms are too weak to resist or by a disturbance of normal physiology and chemistry of the body. . . ."

Pemberton reports that low dietaries were of benefit to certain types of arthritics. He says:

> Certain substances which reach the gastro-intestinal tract through the mouth are carried directly, after absorption by the blood stream, to the joints themselves and can be redetected there almost immediately, in the synovial fluid. Indeed, certain substances, presumably crystalloids, such as glucose, reach a higher concentration in the synovial fluid than may occur in the blood. This observation affords definite evidence that a variety of substances from the intestinal tract have access to the joint cavities. The possibility that deleterious as well as nutritive substances from the bowel may here play a rôle, must henceforth be definitely considered. Freilberg has recently shown that sterile bacterial filtrates injected into the joint cavity of animals may produce changes characteristic of atrophic arthritis.

These facts are of tremendous import. During the past twenty years, the relation of bacteria to disease has been stressed, but the immediate future holds promise of outstanding developments in biochemical and physiologic knowledge of health and disease. Pemberton's work is of special interest to the dental profession. If dietary changes result in a direct alteration of the synovial fluid, why should not one expect to find dietary changes reflected directly in the mouth?

Clinical evidence indicates a definite relationship between the food of the individual and the conditions found in the oral cavity. For instance, we eat candy, and within half an hour find the saliva quite ropy and more highly mucinated; or, we dine "too well," and waken in the morning with a "brown taste." We have eaten nothing during the night, the mouth was clean on retiring, but in the morning we are conscious of a definite change. The mouth tastes unclean, the mucous membrane is coated, and even local cleansing does not entirely remove the condition. In terms of chemistry, we cannot describe exactly what has happened during the night, but one cannot ignore the relationship between the oral condition the morning after and the unbalanced dinner the night before.

May I briefly enumerate instances of food affecting teeth:

1. Biologic experiment has clearly shown that the quality of the tooth tissue and the presence of developmental defects are directly influenced by food.

2. Susceptibility to dental disease is increased by mucinous deposits on the gums and teeth. Thick ropy saliva is associated with indigestion, unbalanced diet or lack of mastication of food.

3. The mouth flora varies with the food débris. Bacteria may be controlled by diet.

4. There would be no acids of fermentation in the mouth without the retention, for some hours, of carbohydrate food débris.

5. The buffer salts and neutralizing power of the saliva depend on the in-

gestion of sufficient of the inorganic salts in the food.

6. The healthy functioning of the masticatory machine depends on the proper selection of foods.

Although much progress has been made in research along preventive lines, I am convinced that there are factors to be determined before we can say definitely that we know the cause of dental disease. Possibly our greatest mistake has been made in looking for a single cause. The problem is not so simple. It is, in fact, most complex, and while as we progress, new factors will doubtless be discovered, our greatest present need is to understand better the things we think that we already know and learn more of their action and reaction one on the other.

The inherent complexity of the problem is due to the fact that the mouth is part of the alimentary canal and is exposed to the ordinary atmosphere (with all that that implies) and yet contains a systemic secretion that reflects varying body conditions. Miller's theory satisfactorily explains the process by which dental caries occurs, but the vital problem remains: Why does decay occur in one case and not in another?

Dental caries is localized at a definite point on the surface of a tooth. What are the laws governing its localization? Why and how is carbohydrate material held in a condition of stasis at one point on the tooth, while the saliva may be flowing freely over the rest of the tooth?

Patients receive considerable information of a general character about food and health, and are asking many pointed questions of their dentists; for instance: Why do my teeth decay though I brush them regularly? Why did that tooth and not this one decay? Do you think my tooth trouble is a question of diet? Or a patient, having read some newspaper or magazine article, may ask even more specific questions, such as: Is sugar really bad for the teeth? Is it true that the body cannot get along without sugar? Is whole wheat bread better for the teeth than white bread?

Surely every dentist should know enough about diet and nutrition to answer such questions, and in language scientifically correct and yet that the patient can understand.

To my mind, it is not a question as to whether the dentist should have a knowledge of food and nutrition, but rather as to whether any dentist can hope to practice modern dentistry without such knowledge.

Within certain limits, the food question may be treated as a mass problem. There is much of public value in general dietary information such as is circulated by departments of health, newspapers, magazines and public speakers. In private practice, when dietary advice is sought by a patient, it is necessary that a more detailed study be made of the individual need in each case.

We recommend that the diet of such a patient be charted, the quantities of each type of food, total calories and the amounts of calcium, phosphorus, iron and other inorganic elements being indicated. It is essential also that there be tabulated the vitamin content, and the average end-product reaction of the foods and their general character from the standpoint of mastication, in order that there be made an intelligent study of the case.

To recommend to every patient a ready-made diet containing one's own favorite foods is to place undue emphasis on the foods themselves and too little on digestion. Neither can we ignore the vital problems of assimilation and metabolism nor the life habits of the patient concerned.

In charting diets, it will be found that, in a given group of individuals, there will often be a wide difference in the foods selected and the quantities consumed, and yet each one of the group, for the individual concerned (considering age, vocation, temperament, climate, etc.), may be partaking of a balanced diet.

Choice of food is influenced by taste, appetite and habit. Food habits formed in childhood frequently persist throughout life. When an individual dietary study is made, these natural tendencies or idiosyncrasies are respected, and we learn whether wrong dietary habits have been acquired. Change of vocation or habits of living, advancing years or what not may point to the necessity for a change in diet, but force of habit may have been so great as to maintain the old diet. A balanced diet, under old conditions, may be quite unbalanced under the new.

In most cases, necessary changes may be made without seriously altering the food habits and are frequently of a comparatively simple character. For example, balance may be reestablished by such changes as the addition of a glass of milk daily, by adding a vegetable or fruit salad or by reducing the daily intake of bread.

Our experience is that, in many studies, comparatively slight changes are necessary to give physiologic balance, but that these changes, though apparently insignificant, are none the less of vital importance. The changes in diet incident to a child's leaving home for boarding school, or a change from one boarding place to another, may be sufficient to convert dental immunity into susceptibility or dental susceptibility into immunity.

Again, loss of teeth, even though followed by the insertion of a denture, may materially affect the health through a change in the dietary habits. The patient may feel that he can no longer eat an apple, enjoy celery or partake of the more fibrous foods. Many such patients have turned to bread, jam and the softer foods, with a consequent impairment in general health. They should be encouraged to persevere and maintain proper dietary habits in spite of their disability.

The charting of a diet must be done accurately and with every article of food included, such as cream and sugar in coffee or tea, butter on bread or toast and dressing on salad. It should always be borne in mind that the problems are biologic and that consideration must be given to variations in digestion and assimilation in different individuals. Figures, therefore, should be considered from this wider viewpoint and used merely as a guide in determining whether the diet is balanced or not.

When a patient is ill, and under the care of a physician, no change in diet should be contemplated without consultation and without the physician's concurrence. In the case of normal, healthy patients with marked susceptibility to dental disease, the dentist is not only within his rights in advising regarding diet, but also cannot be fair to his patient, unless such service is made available.

One might summarize the present status of diet in relation to dental tissues as follows:

1. Local effect of food on teeth.

(a) Dentists are in agreement as to the advantages of eating food of a fibrous character and of a form to require thorough mastication, which brings about healthy function and ample circulation.

(b) Dentists also agree concerning the advantage of foods that are abrasive and have a cleansing action on the surfaces of the teeth.

(c) There also appears to be general acceptance of the value of acid fruits as chemical cleansing agents.

2. Effect of food during developmental period.

Howe, Mellanby and others have shown the effect of deficient feeding on developing teeth in experimental animals. These workers do not claim that a deficiency diet has an exclusive action on the teeth and gums. Indeed it would be difficult to understand how developing teeth could be affected independently of the rest of the body, or, conversely, how a deficiency diet could affect the calcification of bone in other parts of the body and have no effect on the teeth.

Hypoplastic teeth are, generally speaking, more susceptible to dental disease than those that are perfectly calcified. It should be remembered that teeth are sometimes immune in spite of developmental defects, while other teeth are susceptible to dental caries, though anatomically perfect.

3. Systemic effect of food on teeth.

Years ago, the teeth, though an integral part of the body, were treated as though they had little. if any, connection with it. The majority of dentists at that time believed the enamel an inert inorganic mass which, once formed, remained a more or less fixed tissue. There is now a growing belief among research workers and throughout the dental profession that the teeth constantly reflect bodily conditions and that this systemic influence on dental tissues operates not only while the teeth are calcifying but also throughout life. The clinical experience of every practicing dentist shows this to be the fact, but the exact process by which it occurs is not entirely understood. Much more research is urgently needed in this field.

Progress reports in dental research are of great interest to the profession, but it should be kept in mind that few of these are intended for immediate and general adoption. They are intended for discussion, for the creation of wider interest and the stimulation of thought. It is only after such work has been verified, and tentative deductions have been shown to be true, that changes find their way into the recognized methods of dental practice.

To be ultraprogressive and attempt to put into practice at once every suggestion or deduction offered by a research worker would lead only to confusion. Research is usually highly concentrated effort on some one phase of a problem, and we must learn to evaluate the newer knowledge in the light of the old and demand indisputable evidence before we substitute new "theory" for old and tried "practice."

In addition to the direct relationship between diet and teeth, through vital (physical and chemical) forces operating in the mouth, we may all subscribe to the general principle that a balanced diet results in improved health and that this change manifests itself in the dental tissues, just as surely as in other parts of the body.

DISCUSSION

Oren H. Gaver, Baltimore, Md.: This subject is of tremendous importance to the dental profession. The time has long passed when the itinerant tooth drawer performed to an interested audience. Dentistry of today has been placed upon a scientific basis. The public has been slowly educated to this fact, and the medical profession is beginning to be convinced that the mouth means more in the economy of the organism than a place to put a thermometer. I agree with Dr. Seccombe that diet is of the utmost importance in controlling the character of the teeth both during the developmental stages and after the time of their eruption, as to their susceptibility or immunity to decay. We often find teeth which are immune to caries suddenly becoming susceptible, and if we are to render the best service, it is necessary to ascertain the cause

of this change and recommend remedial agents. In order to do this, the dentist should have a thorough knowledge of the vitamin content, ash content and reaction of foodstuffs. If the foods are deficient in calcium salts, the body possesses the power of utilizing calcium from the bony structures, which would include the teeth, to make up the deficiency, in order that the blood calcium shall remain normal. One of the results of vitamin starvation is an atrophy of all the endocrine glands, with the exception of the suprarenals, which play such an important part in the utilization of the lime salts. The diet may contain a sufficient amount of calcium salts, but if they cannot be metabolized because of a lack of the vitamins essential to their utilization, they then become excess material, to be excreted. A knowledge of the reaction of end-products of foodstuffs is essential. The character and reaction of the saliva usually serve as an index of the type of food ingested. A rich carbohydrate and protein diet leads to acidosis, which is reflected in the saliva's giving to the teeth an unfavorable environment for maintaining their immunity to disease. Acids are continually being formed in the body owing to the breaking down of the body tissues. These cannot be controlled, for they are physiologic changes. The acids formed from the foodstuffs can and should be controlled as they are precursors to strain, irritation and disease. If we look into the chemical changes that foods undergo, we can readily understand how those foods which are acid to taste or litmus, such as the citrus fruits, become strong alkalinizers. The organic acid is quickly oxidized and disappears as carbon dioxid and water, while the alkaline mineral remains as sodium, potassium, etc., to unite with other substances to form salts, principally as carbonates, which are the strongest buffers of the blood. Dr. Seccombe has touched on a vital point in referring to the possible influence chemical toxins have on the body as causative factors in diseased conditions. Owing to the advance in knowledge of dietetics, we can readily understand why so many foods are incompatible. They cause a derangement of the gastro-intestinal tract, thus preventing the foods from being digested and absorbed. The foods then become the object of attack by the bacteria, being converted into products of fermentation and putrefaction. The toxins thus produced, unless promptly eliminated from the intestines, are absorbed and crowded into the excretory system. This produces irritation and inflammation of the tissues, and following the decrease in vitality, they easily fall prey to microbic invasion. The foulness of breath and coating of the tongue referred to in the paper are due to the elimination of these toxins through the saliva and exhaled air. This influences the character of the saliva, making it an excretion instead of a secretion, and thus an unfavorable environment is created for the teeth and supporting structures. The practicing dentist of the future will be one conversant with the biochemical and physiologic changes that the food undergoes from the time it enters the body until its waste products are eliminated as excreta. He will also be fortified with a comprehensive knowledge of what constitutes a balanced diet suitable for all classes of individuals. I agree with Dr. Seccombe that no condition can be attributed to dental disease alone, but rather is a complex problem, including the diet, endocrine system, nervous system and the character of the digestion. If any one of these are interfered with, the effect will be manifested by a deranged activity on the part of the others. Dental colleges, in the future, will stress the importance of physiologic and biochemic changes in the body more than in the past, thereby emphasizing that an ounce of prevention is worth more than a pound of cure.

J. Paul Wintrup, Wilmington, Del.: Dr. Seccombe has presented a group of ideas which may presage the future control of all dental caries. Remarkable progress has been made during the past score of years in the treatment of oral disease. We have developed excellent remedial agents. Early tooth decay, if properly handled, is not so much of a problem today, if a sufficient extension of all cavities is made to avoid recurrence. Dr. Howe has given us the silver nitrate reduction method for the prevention of occlusal decay. Interproximal and gingival caries remains to be mastered. The control of dental caries must begin at the time of conception or as soon as it is known to have taken place. The problem of diet must be considered from then on, during pregnancy and after parturition, for two chief reasons: (1) to avoid developmental defects in the teeth of the child; (2) to avoid destruction of the mother's masticatory apparatus. Pregnancy, more than any other condition, demands a specific diet and an abundant supply of calcium. For some

time we recommended, besides a balanced diet, a solution of hypophosphites. It is good, but we believe that there are better agents available. We are following two courses now: 1. For one-half of our patients, as soon as we learn of pregnancy, we prescribe 8 drops of ergosterol a day and 3 tablespoonfuls of cod-liver oil a week. 2. For the other half, 3 gm. of normal calcium gluconate every day and 3 tablespoonfuls of cod-liver oil a week are prescribed. I believe that calcium gluconate will give better results because the calcium is in a more usable form, but I know that the other is satisfactory. Our evidence indicates that fact. The calcium and vitamins A and D are added to the prescribed balanced diet because we believe that the mother cannot get enough of the required elements in the foods alone. It is possible to change osseous structures by changing diet. The work of A. Y. Russell, of Baltimore, the greater part of which I am sure has not been published, describes this fact beautifully. Dr. Russell, using the roentgen rays, with all factors under control, i.e., position, exposure time, developing time, etc., has shown that a jaw, porous and unhealthy in appearance in the beginning, may be made, by administration of certain foods, more dense and normal in appearance. He can even make it more dense than normal and then reduce the density. At least, the roentgenograms indicate this by their appearance. If this is possible in mandible and maxillae, it should be possible at least in the dentin of a tooth. As for the enamel, we should have to believe in the theory of circulation in that structure, and I am not ready to accept it in full. This theory is important because, by believing in it or not believing in it, we can arrive at the conclusion that caries has either an extrinsic or an intrinsic etiology. Either the lymph, shall we say, or the saliva, if either, carries the destroying element! There is some room to question the presence of ameloclastic cells. Yet we might find some substance which will affect the dissolution of the interprismatic matrix. All of this does not account for plane surface decay, in my opinion. It is, I think, altogether an extrinsic action, dissolution of the enamel by acid. And I lean toward the opinion that almost all of the acidity is from the decomposition of carbohydrates. Chemical analysis of specimens of saliva show relatively little variation in p_H. It is a buffer solution. The value of cleanliness is, hence, even though we consider diet and good dentistry, still highly important. I believe, like Dr. Seccombe, that highly abrasive foods help considerably in producing this result. Some of you may feel after Dr. Seccombe has talked so extensively about bacteriology and pathology that he places them secondary to diet. I do not believe that he does, but in his enthusiasm over the food factors, he has said much of those subjects. For instance, his paper states that bacteria have even been found in the blood stream. Bacteria are not found there under normal conditions and unless phagocytes or antibodies destroy the organisms, disease will ensue; if not from virus actually secreted by the bacteria, then from their decomposition. I believe the work being done to differentiate types of arthritis and other infections to be a very valuable one. There is no doubt that all of the conditions attributed to focal infection have been exaggerated. We are faddists. Yet some conditions are rightly laid to focal infection. This is not conjecture. The evidence can be produced. Dr. Seccombe states that there appears to be a general acceptance of the value of acid fruits as chemical cleansing agents. This is true. I wish to bring this point to your attention with the permission of the worker, who has not reported his results as yet: Extracted teeth half covered with wax were placed in solutions of various fruit juices. The teeth were examined in twenty-four hours and the exposed portions were highly decalcified. I wonder if, in our effort to obtain vitamins, we are injuring the teeth with these fruit juices. In considering the etiology of dental caries, it is important to look at all aspects. Diet is unquestionably a big factor. Habitat is another one, because it controls diet. I am convinced, moreover, that heredity plays a very definite rôle. In balancing our diet, so as to provide the proper foods, we should not lose sight of the necessity of providing a large amount of good water and a similar quantity of fresh air and sunshine, particularly sunshine, natural or artificial. Dentists must realize the value of ultraviolet light. It is a tremendous factor in the metabolism of calcium, our king of elements. Dr. Seccombe's idea of diet analysis may appear, at this time, to be difficult of accomplishment. Yet, as we progress, it will not be so, because our time will be spent more in preventing the conditions to which we are now devoting hours in an effort to return to a pseudo-normalcy.

Dr. Seccombe (closing): One suggestion that came out in the discussion was the importance of the prenatal treatment. I quite agree with that, both from the standpoint of proper development of the teeth and from that of the mother herself. We must think of the developmental period as extending from three or four months before birth, up until 8 or 9 years of age, because the crowns of the teeth are calcifying during that period. It is a question of not only the prenatal but also the period of lactation, of the first year after birth and, following that, the preschool age period, and that of childhood, up to about 10 years of age. This is the time that is most important from the standpoint of the development of the crowns of the teeth, rendering them more or less susceptible to dental caries. The suggestion was offered that it was impossible to give an expectant mother a diet containing a sufficiency of the bone forming element. I must disagree with that statement. I believe that it is quite possible. A few years ago, we carried on a series of experiments in one of the hospitals in Toronto, taking the saliva of expectant mothers and analyzing it from week to week until the time of delivery, and found that, in a group of these cases, there was a gradual loss of calcium and phosphorus in the saliva of these women living on hospital diet during pregnancy. We then changed the diet and continued to make chemical analyses of the saliva, and found we could put back into the saliva, through diet, the normal amount of calcium and phosphorus. From these experiments, as well as many other dietary studies, I believe that we can no doubt, through a proper selection of food, supply to every individual (even during that period when there is an unusual demand for calcium and phosphorus) a sufficiency of all the vital elements that are necessary. In other words, it is not necessary for us to resort to other than natural foods to supply these elements normally through the diet. To say that everybody must supplement the diet of natural foods with certain other products is, in my opinion, not the case. It is quite possible to supply all of the necessary elements through a proper selection of natural foods—a balanced diet. We have to face, after eruption, the question of caries, which introduces local conditions; bacteria, food débris, mucin, saliva and other externals. All of these are related in one way or another to food and diet. As members of the dental profession, we should give more study to this subject so that we may be able to render a better dental health service to our patients. The foods that we used in the corrective diets of these hospital patients were fruits and vegetables high in calcium and phosphorus; and milk, which is the highest of all. That is the food that Nature elaborated for the first year of life, when so much bone formation is going on. We find that there is no other food so high in calcium and phosphorus. Figs are also very high in calcium and phosphorus, and so is cheese. Of course, it is not recommended that all of these foods be given to a patient in unlimited quantity.

THE PARTIAL DENTURE VS. FIXED BRIDGEWORK WITH SPECIAL REFERENCE TO THE TREATMENT OF INVESTING TISSUES*

By THOMAS B. HARTZELL, M.D., D.D.S., F.A.C.D., Minneapolis, Minn.

ONE frequently hears partial dentures vigorously criticized for one reason or another; and it frequently occurs that the individual pronouncing the criticism will speak highly of the advantages of fixed bridgework in preference to partial dentures as a means of restoring teeth that have been lost by extraction.

The decision to use one form of restoration or another should rest essentially upon the fundamental needs of the patient in each case, and certainly not upon prejudices of one sort or another. To me, these prejudices as I have encountered them are based upon ignorance and a difference in skill in the prejudiced individual in the two departments of restorative work—just dislike of one type of work, at the expense of another. Certainly, if a dentist can build a fixed bridge that will endure and give service, and habitually experiences failures with partial dentures, he will lean toward fixed bridgework as a restorative measure.

Not long ago, I listened to a sharp criticism of partial dentures, made before one of our state societies by an advocate of fixed bridgework. The discussion betrayed an absolute lack of knowledge as to why teeth are lost, facts which should be common knowledge of the whole dental profession in this day and age, though, unfortunately, they are not, because dentistry, as every other walk of life, contains a certain group of individuals who refuse to see the truth even though it be self-evident. This vigorous advocacy of fixed bridgework and condemnation of partial denture work was based chiefly on the alleged fact that the teeth which carried clasps invariably decayed long before the remaining teeth commenced to decay, and that this decay was caused by the clasp; a most foolish and an untrue statement, betraying the fact instantly that the man did not know what he was talking about, and further proving that he had never taught his patients the fundamental truth that all patients should be taught in the first contacts with them, namely, that decay of the teeth is the result of bacterial action, that true decay never has been known to occur except in the presence of multitudes of living organisms.

There is nothing in clasp metal to dissolve enamel. Confine a mass of bacteria against enamel under a clasp, and, of course, the enamel will be broken down rapidly by the bacterial solvents confined under the clasps. The dentist should teach the patient to keep clasps clean, removing these bacteria from the clasps and teeth daily, morning and evening.

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This knowledge, of course, this man could never give to his patient, because he either did not possess it himself or he did not believe in its truth. It was probably true that his patients had suffered from vigorous and rapid decay under the clasps of the teeth which he had covered in his partial denture work, and he had finally given up partial denture work because of this continuous failure of his partial dentures by reason of decay of the teeth covered by the clasp.

I have had partial dentures in use in my practice more than thirty years, and my patients do not experience decay under the clasps that retain these partial dentures, for the simple reason that I teach every patient to *see* the bacterial masses that accumulate on his teeth and the inside of the clasps by supplying him with disclosing stain, which he applies for himself to test whether his teeth are reasonably free from bacterial life. I expect him to splash disclosing stain on his teeth twice a week, and I request and expect him to scour off everything that is revealed by stain and to retest his operation by restaining to see whether or not he did an honest piece of cleaning. In addition to this teaching, I carefully explain that a mass of bacteria gather under the clasps, a much greater mass than could accumulate on an exposed surface, because the bacteria grow on the inner surface of the clasp, too, and the greater amount of bacterial acid capable of dissolving enamel is protected from dilution by saliva by the clasp itself, so that decay of the tooth's surface will go on more speedily under the clasp than on the surface of the teeth, which are exposed to the cleansing action of the cheeks, tongue and lips, and the action of mastication. Therefore, in addition to teaching each patient the technic of getting reasonable freedom from bacteria, we should insist that he clean the inside of the clasp with an orangewood stick whittled to the right shape, scouring the clasp until it is as brilliant as when first made.

For the last eight or ten years of my practice, I have spent more and more time in instructing patients as to just how to attain a moderate degree of freedom from bacteria on the surfaces of the teeth. Several years ago, I began furnishing each patient with a box of toothpicks for tooth cleaning, teaching the patient how to push the pick carefully between the teeth until it wedged, so that it would break up the bacterial coat accumulating on the proximal areas. Thousands of teeth are made to bear bridgework which are better adapted to clasps and partial dentures than to bridgework, and many of these thousands of teeth have had their pulps gradually destroyed because of the severe grinding necessary to place crowns to fit accurately at the necks of these teeth. The damage caused by the acid of the cement which coagulates the fibrils of the odontoblasts in the newly ground dentin, often ground close to the pulp, leads to the death of the pulp and the loss of the bridge itself, whereas, had partial dentures been skilfully adapted to the case, and the patient taught to keep his teeth and the clasps clean, these teeth might still be comfortable and healthy. Certainly, fixed bridgework has a valuable place in our restorative work, and will never be discarded, but partial dentures, skilfully adapted, are often much to be preferred to fixed bridgework. The ease with which a partial denture can be removed and all its surfaces, including the inside of its clasp, cleaned and the greater ease of cleaning the bacterial growth of the day from all the remaining teeth, makes the partial denture operation the operation of choice. We must keep in mind, with all denture

work, that teeth change position from hour to hour, from day to day. The bone of the jaw also is in constant change, and all partial dentures should be and can readily be readapted to the surfaces from time to time as these changes take place.

The dentist who fails to teach his patients one of the important things that he should teach them, namely, that the teeth are in more or less constant movement and that the jaw and alveolar process are constantly changing, fails to take advantage of one of the most valuable facts that he can convey to his patients, which will explain to the patient why it is necessary to see his dentist regularly, that these changes, as they occur, may be compensated for by readapting the denture in such a manner that occlusion is kept perfect. If we all observed some of these simple precautions, our contacts with the patient would be much happier for us both, and a reasonableness of judgment would be developed in the patient that would prevent his making many of the unjustly harsh criticisms of the dentist's work which one frequently hears; which would never occur if these simple facts regarding mouth hygiene and physiology were effectively taught the patient. We should constantly keep in mind the fact that most of the cases of faulty and imperfect occlusion are the result of disease-producing bacteria growing and accumulating on the sides of the teeth. The bacteria masses adjacent to the gums destroy peridental membrane fibers, and teeth released from their normal anchorages begin to move. The smashing power of the muscles of mastication furnish the force to drive these teeth in one direction or another, thus producing traumatic occlusion, which could not have developed had no peridental membrane fibers been destroyed by the ferments of pus-forming bacteria. In fact, the need for partial dentures and fixed bridges arises chiefly because of the loss of teeth by decay and pyorrhea, both diseases produced by bacterial products. I again emphasize the fact that the choice of a fixed bridge or a partial denture to compensate for teeth lost by bacterial destruction should be decided on the basis of whether we can prevent further bacterial destruction of the teeth and alveolar process that remain.

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DISCUSSION

Herbert E. King, Omaha, Nebr.: The choice between possible types of restorative work is determined by so many things that a careful survey and analysis must be made in justice to both patient and dentist. In order to outline the field clearly, it seems that we should state our definition of a partial denture as any appliance depending for the major portion of its support on the mucosa and not on the teeth. This in contrast to any bridge either fixed or removable where the teeth must carry all or most of the stress. Most partial dentures then fall into the class which have progressed past the possibility of fixed bridgework. The cases evidently under consideration in the paper would include the smaller removable bridges or a case of several spaces of one or two teeth that can be restored with a removable bridge, the cutting of many abutment teeth being avoided. In these cases, the choice must be made. In many very "short bite" spaces, it is almost impossible to make a fixed bridge which can be kept even reasonably clean. As emphasized by Dr. Hartzell, the ability to keep a clean mouth is all important, and in these cases in which close occlusion prevails, it would be a deciding factor in choosing the removable bridge. It is true that there are dentists who make only one type of restoration and condemn all others, but that is simply beyond our control. It seems to me that a most important element is the patient. We all, I am sure, have patients to whom anything that must be taken out of the mouth will prove a nuisance. He is either very nervous in his makeup or very careless, and

almost any fixed piece will be much more satisfactory than anything removable. He is the type that will "forget" to wear the appliance, and it will never be quite comfortable. He will of necessity become accustomed to the bridge that is cemented in place. Then, there is the patient who seems intelligent but on whom education of a dental nature seems lost. It may be the fault of the dentist, but we all meet the patient who will not cooperate in the care of the mouth. The ideal patient will do as his dentist suggests. It seems that in emphasizing the clean clasp, Dr. Hartzell should also place importance on the properly formed clasp. The fact is too well known to this audience to be dwelt on in detail, but there is the old type of clasp which covers many times the amount of tooth surface that is necessary. It is indeed a matter of surprise that there is not more caries rather than that we have it at all. A properly made clasp, be it a cast clasp, wrought wire or any other type, should have contact with only the small enamel surface in about three points on the tooth. Such points can be selected for each individual clasp to do just the thing that the clasp is expected to do and, at the same time, offer little opportunity for caries. Then, after the clasp is correctly designed, it may easily be kept clean. The Roach bar clasp type represents a long step toward lessening the chance of decay. I would question the crowding of toothpicks into interproximal spaces. It will work nicely many times, but there is again the careless patient or the one with an apparent lack of intelligence who will injure that most valuable tissue, the gum septum. The choice of the appliance, then, is determined, by many things: the patient, the dentist and his ability to restore lost organs with as little damage as possible.

SURGICAL TREATMENT OF CHRONIC MAXILLARY SINUSITIS OF ORAL ORIGIN*

By HENRY SAGE DUNNING, B.S., M.D., D.D.S., F.A.C.S., New York City

MAXILLARY sinusitis of oral origin has become quite a common disease of late years on account of the great improvement in our methods and technic of making oral diagnosis. A great deal of credit in our improved diagnosis of these cases must go, I think, to our improved skill in taking and understanding roentgenograms. Notwithstanding our readier recognition of diseases of the antrums, I believe that the maxillary sinus is still far out in no man's land, between the front lines of the rhinologist and those of the dental oral surgeon.

At the outset of this paper, I should like to make a plea for closer cooperation between the nose and throat specialist and the oral specialist. Having worked about five years of my life in a large nose and throat hospital, I can say positively and without fear of contradiction that it would be a fine thing for every dental man who has a flair for oral surgery to be connected with a nose and throat clinic in some capacity for a certain length of time. The experience gained by him in working with the rhinologist will be most profitable, as he will obtain by this connection a working knowledge of the anatomy and the physiologic function of the nose and its accessory sinuses. Without this knowledge, the dental surgeon ought to avoid

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treating the maxillary sinus as nearly as possible.

Having taught oral surgery to postgraduate dental students for over twenty years, I have learned by experience that few dental men have much idea about the important cavity that we are discussing, probably because the subject is poorly presented in the dental school.

Many dental men get into trouble at times when a tooth or root is forced into an antrum; when there is a postextraction infection, or when a slight fracture of the antral wall occurs in the removal of impacted third molars, in an osteitis of the maxillary bones. They do not seem to know how to drain the antrums, when to pack and when to sew up, and, most important of all, few seem to understand how to get a root or a tooth out of the sinus after it has been unwittingly forced into the cavity.

I shall cite two cases of maxillary sinus complications which occurred during the past year. Both were serious and both could have been avoided quite easily had the operator had a little more knowledge and a little more courage.

In the first case, a very busy obstetrician went to a young dentist for the extraction of a pulpless upper second molar. The palatal root was fractured in the extraction and when an attempt was made to get it, the root was forced into the antrum. The operator then made a large opening through the alveolus and several attempts were made to remove the root, but to no avail. The wound and the antrum were packed fairly tightly and, in three or four days, the case was referred to me. The doctor had been suffering excruciating pain, had had little sleep and, to use his expression, was "all shot to pieces." I removed the packing, which was none too clean, and a considerable amount of pus came down to the opening from the antrum. I irrigated the antrum and took an anteroposterior plate of the head and also a dental film and located the root quite easily. I then turned back a large flap, extending from the alveolar ridge over the anterior wall of the antrum, and made a large opening through the anterior wall of the antrum. In a few minutes, with the Coakley antral curet, I removed the root fragment and closed the wound to the alveolar ridge. The patient still had a large opening through the alveolus of the extracted tooth. A maxillary infection was developing, which proved very persistent. As the patient was very busy and therefore very irregular in his visits to the office, he still has an opening into the antrum from the original wound and he still has a sinusitis that lights up now and then, in spite of nasal drainage and other methods of treatment.

This case developed last March, and for weeks the patient suffered and lost much time from his practice. He still has a troublesome sinus.

In the next case, a young man had had a second molar extracted. There was no third molar present. When the dentist attempted to extract the second molar, he found the case stubborn, but he was stubborn and *strong,* and so won out. However, he fractured the tuberosity of the maxillary bone, then grew frightened and thought it best to remove the fractured bone which consisted of the entire tuberosity. He then thought it safer to pack the antrum tightly with gauze. The dentist sent the patient to me in about a week. The antrum and the soft tissues were infected; the packing had forced the edges of the wound widely apart, and I could almost put two finger tips into the antrum. It took some time to clean up the tissues sufficiently for plastic

closure, which successfully terminated the antral condition.

These two cases illustrate points that I wish to bring out later.

For many years, the rhinologist has felt that the maxillary sinus was his property because he has considered that from 70 to 90 per cent of all maxillary sinus infections descended from the frontal or ethmoid sinus or from the nose itself. I believe that the majority of nose and throat men today feel that there are more ascending infections from the mouth involving the antrum than were formerly recognized, but that still the larger percentage of infections of the maxillary sinus come from above downward. Most of my friends among the oral surgeons, I think, feel that the percentage of antral infection of oral and that of nasal origin is much nearer 50-50. We, of course, might say that the oral surgeon sees more of these cases originating from the mouth, and that the rhinologist sees more springing from the nasal accessory sinuses. In these days when so many patients are having teeth roentgenographed as a matter of routine, latent antral infections are discovered, and many teeth are removed whose alveoli enter the antrum on gentle curettage. On the removal of many of these teeth, it is found that there is a chronic low-grade infection in the antrum, which has been draining comfortably through the middle meatus for months. This mild infection has taken care of itself and the condition has never become acute. We are today giving much thought to the periodontal structures, to the maxillae and to all the tissues of the oral cavity. We are ever on the lookout for bone cysts, neoplasms, infected areas, etc. Few teeth today are extracted by the careful practitioner without being roentgenographed first. The condition of the bone is known and the bone is often curetted and rongeured even though the antrum is opened.

I have had many cases during the past years in the hospitals and in private practice in which I have had to make a wound in the maxillae that has placed the mouth in communication with the antrum. In many cases, the antrum has been found to be infected.

When the operator has from force of circumstances made a good sized oral opening into the antrum, and has found the antrum diseased, there are two main factors in the subsequent treatment which should appeal to him: (1) cure of the antral disease as soon as possible, and (2) closure of the communication between the mouth and the antrum at an early date.

If we study the physiologic function of the maxillary sinus, and also its anatomy, we find that the antrum was intended to communicate normally with a comparatively clean cavity, the nose; that the nose and the antrum are lined with the same membrane, and that about the same bacteria are found in these two cavities, the nasal and the maxillary sinus. It is evident to the casual observer that Nature never intended the mouth and the antrum to communicate, and never intended the mixing of their different bacteria and secretions.

It has been proved clinically that a permanent opening from the mouth into the antrum may result in an infection ascending into the maxillary sinus, later causing a pansinusitis; yet how often do we see the antrum, in these chronic infections of oral origin, irrigated for weeks through the opening in the mouth and packed with gauze? Why should a large oral opening into the antrum be permitted to exist? The mechanics of this treatment are wrong. The operator is very apt to carry infection from a dirty

cavity, the mouth, into a comparatively clean cavity, the antrum. Dentists in the past have, I believe, caused a great deal of trouble by persisting in this old-fashioned method of treatment. A gauze drain inserted into the antrum from the mouth often draws up secretions from the mouth by capillary attraction, and frequently reinfects the cavity one is trying to cleanse. Until a few years ago, textbooks were advocating the extraction of a tooth to permit treatment of a maxillary sinus infection. These books did not specify removal of a diseased tooth that was causing the antral infection, but a healthy one, for drainage purposes only. The sacrifice of a healthy tooth in the old days meant nothing, and the establishment of the communication between two cavities containing different secretions and bacteria did not seem to worry anyone. This method, known as Cowper's, is not advocated now, although it is often mentioned.

The etiology of maxillary infections in the past has not been carefully considered. Not long ago, a well-known rhinologist referred to me a case of maxillary sinusitis in which he had just performed a radical antral operation. I was told that there was "a sort of 'gum boil' over the second bicuspid," and was asked to treat it. My roentgenographic films disclosed a decided shadow extending to the antral boundary over the second bicuspid, which was a pulpless tooth. Under procain, I removed the tooth, and the curet entered the sinus very readily. On exploring the so-called "gum boil" above the tooth, I found that it led to necrotic bone. Most of the alveolus of the offending tooth was removed, and an area of dead bone as large as a penny was curetted from the anterior wall of the antrum after a flap of the soft tissues had been released. I do not believe that this case was properly diagnosed before the first operation. There was some diseased membrane over the end of the infected bicuspid, and I am quite sure that the antral infection would not have cleared up in many months, if ever, without some surgical treatment of the infection of the alveolar process. The type of antral infection should be diagnosed early, before treatment is begun, and this cannot be done accurately without taking oral roentgenograms in conjunction with the anteroposterior plates showing the sinuses. If the infection is a descending one from the frontal or ethmoid sinuses and the nose, the case generally belongs to the rhinologist. In some cases, it can be handled by the oral surgeon, if he has had training in nose and throat surgery. If the infection ascends from the maxillary bones and teeth, it cannot be cured by the rhinologist alone, unless he is familiar with the mouth and dental structures. In either of the foregoing cases, the cause of the infection must be removed; and if the case is chronic, the antrum must be cleaned by gentle curettage and must be irrigated, and drained properly. In nearly all of these cases, the sinus should be irrigated with a warm saline solution through an opening under the inferior turbinate, and if the infection is of oral origin, the washings drain downward into the mouth. If the case is not of oral origin and there is no opening into the mouth, the washings naturally return to the nose and are discharged through that cavity. Cornelius Coakley, of New York, says that there is too much sinus surgery, that nearly all cases of maxillary sinusitis can be cured by irrigation and ventilation. I do not see why the mouth opening in the oral cases should be packed, in any case, for more than a few days, even if suppuration has not ceased and although the tissues around the oral open-

ings are not in sufficiently healthy condition to bear sutures. In many cases, it is not advisable to close the oral opening mechanically for a time on account of suppuration. In these cases, the mouth and the maxillary sinus should be separated by a wall-fitting "saddle-plate" that does not press at all on the edges of the wound leading into the antrum. No plug or projection that enters the opening should ever be used. I cannot condemn too severely the use of any appliance that tends to establish a permanent opening between these two cavities. This plate, which is best made of rubber, covers the labial and palatal surfaces of the alveolar process, and is generally held by clasps. Of course, this is temporary, and is to be worn only until the suppuration has subsided and the plastic repair of the opening can be accomplished. This plate, in a way, acts as the valves of the heart. It allows of drainage from the antrum downward, under the plate into the mouth, but prevents seepage upward of the oral secretions into the maxillary sinus. These plates, if constructed properly, never prevent drainage, and they keep the antrum cleaner than any gauze drain possibly can.

In searching the literature, we find little mention of any operative procedure for closing off an opening from the mouth into the maxillary sinus. Little attention has been given to the fact that the mouth and the antrum are two very unlike cavities, that their secretions and their bacterial content vary greatly, and that they are much better neighbors when they are separated by a barrier of healthy tissues. In cases in which the opening persists between the two cavities, the only permanent method of preventing communication is by means of a plastic operation. I have closed many openings into the maxillary sinus by undermining the soft tissues around the edges of the defect, and then drawing the tissues together. This procedure will suffice in some cases, but, in the majority of cases in which the opening is of considerable size, this method will fail, as the sutures will slough out, because of tension. Dean has described an operation for closure of the opening between the mouth and the maxillary sinus. He does not make very clear the arrangement of the flaps, but explains in detail his method of suturing and the materials used.

Infections of the maxillary sinus are often not diagnosed by the dental surgeon and are frequently inadequately treated. After all that has been written on this subject, it is disheartening to pick up current articles in which the author advocates the use of the rubber plug, and, further, to read that "the plug in the antral opening helps to hold the plate in place."

I once had a patient in a clinic who had suffered for years from a chronic maxillary infection. He had been treated in Vienna. What the early treatment was in this case was vague, but for thirteen years he had been wearing a plate with a large plug projecting into the antrum. The condition of that antrum was remarkable. The cavity was almost obliterated by thick polypoid tissue, and, needless to say, it was badly infected. The lining membrane was more like leather than like normal tissue, and often the patient had to relieve conditions by trying to irrigate through the opening himself. He always carried a syringe with him, he told me. It was difficult to clean out this sinus, after it had been reinfected by the mouth for thirteen years, and the closure of the opening into the mouth was not attempted.

For the closing of oral openings into the maxillary sinus, I advocate the oper-

ation described below. I do not claim that it is original with me.

TECHNIC OF OPERATION

A flap operation was found best, after several failures in trying to bring the tissues together. In closing over large openings, I found that a flap from the labial tissues was not very satisfactory, in that it was difficult to get enough tissue at times, and also that the tissue obtained was not very strong. The palate was turned too early in my experiments, and I am not sure whether I adopted the idea at Queen's Hospital, England, during my three months' stay there. It is now the only flap I use, and I feel that it is the most efficacious one. The tissue is thick and tough, and pares off evenly and easily. A palatal flap is taken, extending from the median line at the junction of the hard and soft palates anteriorly to about opposite the bicuspid teeth. The flap is thoroughly separated from the palate down to the periosteum, but the latter is not disturbed, as this allows for better repair by the formation of granulating tissue in the defect caused by the sliding of the flap outward over the opening that is to be closed. The flap just described is very thick and, of course, is well nourished, as it has in its base the anterior palatine artery. The next step is to free the labial flap, including the vestibule of the mouth, and to free the opening to be closed of all soft tissue down to the bone. Any sharp edges of bone must be smoothed off, and often the alveolar process trimmed, so that the flap will more than cover the opening without any tension, when sutured to place. The labial soft tissues are now thoroughly undermined and freed, often upward to the extent of an inch or more on the anterior surface of the superior maxillary bone. The periosteum is not disturbed here either, and a curtain of soft tissue is dropped to meet and overlap the original flap from the palate. Sometimes, the anterior border of the labial curtain just dropped is freed so that a labial flap thrown over the palatal flap can be tucked under the foregoing labial curtain, or can be sutured over the curtain after the latter has been sutured to place over the bony opening to be closed. It does not matter much which way one disposes of these flaps, if they are made to overlap each other like a double breasted coat and sutured securely. It matters little what material is used in suturing. I have used silk, horsehair and other materials, but now prefer the dermol suturing material, artificial silk. The secret of success in this operation, as in all plastic operations, is to have plenty of tissue, no tension and a good blood supply. I have not in any one of these operations had the opening into the antrum fail to close, and it has become with me now the routine method of closing these annoying openings between the maxillary sinus and the mouth.

CONCLUSION

I should like to make a plea for better diagnosis in these cases; to urge that drainage through the nasal route be used in the majority of cases, and that the opening between the antrum and the mouth be closed as soon as possible.

DISCUSSION

W. L. Shearer, Omaha, Neb.: It seems to me there is a better approach to the maxillary sinus in operating for maxillary sinus disease than the one just presented. For many years, I have criticized the approach to the maxillary sinus. The Denker incision, I think, should be positively condemned by all who appreciate normal anatomy. If one will employ a flap extending from the lateral or central incisor about one-eighth inch above the cemento-enamel junction of the teeth, passing backward and up over the tuberosity, then, if

necessary, making a short incision at about the position of the central incisor, extending upward to where the mucosa folds upon itself, perfect access can be attained and severing of the nerves, veins and arteries, which are destroyed by the majority of other incisions, be avoided. With this type of incision, postoperative complications, such as cicatricial contraction and numbness of the nose, are avoided. If one is not going to remove the membrane, I can see no reason for using a curet, as a general thing, because a sinus can be cleaned perfectly by the use of a sponge with the selvage edge turned in and that has been wrung out of hot salt water. In this way, there is no injury to the ciliated mucous membrane of the maxillary sinus. Further, when one has passed a trocar from the nose into the maxillary sinus, the traumatized schneiderian membrane, as well as the mucous membrane of the sinus, should be picked up with a hemostat and removed with a sharp knife. The bony edges of the opening from the nose into the sinus should be made as smooth as possible. Ordinary irrigation with physiologic sodium chlorid solution will be sufficient. An old surgeon for whom I performed a maxillary sinus operation many years ago asked me to saturate iodoform gauze with sterilized olive oil before placing it in the antrum. I have been very grateful to him all these years for the suggestion, because, with this procedure, I have been able to remove the dressing either the first or second day after the operation without undue disturbance to the patient. Since I have learned to know the importance of the operation known as alveolectomy and partial alveolectomy and have had an opportunity to clean up many infected mouths, I am trying to assist my brothers in the profession with sick patients. I have found that the majority of maxillary sinuses are infected at the time the teeth are removed by blind curettement. The majority of infections of the maxillary sinus that are of dental origin are not primarily of dental origin, but are secondary to the removal of a tooth whose infected root tip is in close proximity to the floor of the sinus.

Samuel S. Sobell, New York City: I would like to ask Dr. Dunning a question in reference to the infected vital pulp that seems to affect the condition of the sinuses; and which rhinologists find difficulty in clearing up. We sometimes find this condition in second bicuspids when the first molar has been removed, leaving a residual area; and we sometimes find infected vital pulps in other teeth in which inlays have been used. How would the dentist diagnose an infection of a vital pulp, and predetermine such a condition before he extracts the tooth?

J. E. Nyman, Chicago, Illinois: Dr. Dunning made reference to a case which I saw in consultation with Dr. Sahad at Palm Beach and which emphasized the importance of removing root ends in a careful surgical manner, and not merely doing it by simple application of force. Furthermore, it emphasized the necessity of closing these traumatic openings of the maxillary sinus that have occurred by making flaps with a very broad base to insure good circulation, and doing it immediately. Undoubtedly, failure of the first effort to close that traumatic sinus (which was not the work of Dr. Sahad) was due to the fact that the operator did not provide for flaps or afford sufficient basis for circulation, and there was also too much traction made in suturing the edges together. I think most of the cases of maxillary sinusitis that arise from tooth infection are of the unfortunate type that are apt to develop granulations in the membranes of the maxillary sinus, just as we have granulation so often about the apices of infected teeth; whereas, the infections that occur through the nasal cavities themselves are not so apt to result in those polypoid growths. Once they have developed there, it is practically impossible to do anything more than relieve acute conditions by an operation through the side wall of the nose beneath the inferior turbinate. It is imperative that once a tooth root has become dislodged and located in the antrum, we acquaint the patient with the fact immediately and medicolegal experts tell us that our chances of emerging successfully from a possible damage suit are much greater provided we can prove that we immediately acquainted the patient with the fact that something had occurred that was unlooked for and could not be prevented, that it had occurred before, and would occur in the future. At the same time, we should immediately call in a good nose and throat specialist if we have not a good oral surgeon. The only wise thing to do is the canine fossa operation. Then we can see the whole maxillary sinus from front to back, top to bottom, side to side. Sometimes, small septums will come up through the floor and a

tooth will have lodged where we cannot see it, even though we may locate it in the roentgenogram. When we attempt to go in from any other point, we cannot find that tooth and therefore cannot remove it. In cases of chronic maxillary sinusitis which undoubtedly have been complicated by some tooth infection, this granular deterioration of the membrane of the maxillary sinus will frequently progress upward and on the inside until the final involvement is of the wall of the posterior ethmoid cells. This can be definitely avoided only by the canine fossa operation. I have operated a great deal in conjunction with Dr. Sahad, and he is one of the best operators on the sinus that I have ever found anywhere, and over and over again, when we have succeeded in cleaning out all of the mass of polypoid infected material and infected granulomatous tissue (most of it can be removed by swabbing with coarse gauze but some of it has to be gently curetted with a ring curet), if we throw a light and illuminate the whole cavity, with a very fine cannula we can safely enter the ethmoid sinus and it is surprising how often a secondary infection will be found. A side wall window into the nose is made, but the opening through the canine fossa is immediately closed by suturing. I want to emphasize what Dr. Dunning has said—do not pack a maxillary sinus opening into the mouth; let it close up if it will, and as quickly as it will.

Dr. Dunning (closing): If we could go away with the idea that we are going to operate through the canine fossa, and that when there are roots in the antrum, we are going to get them out and not let them go to a later date, it would be well. What I mean is that the young patient I referred to was sick. He had been out of work about six weeks and had suffered a great deal. He was a nervous type and kept putting off a radical antrum operation, which he needed badly. The poor young dentist who broke off the root in the antrum could not get it out. If a root does get into the antrum, and you haven't a sufficient opening in the maxillary sinus, go through the canine fossa and make a large opening. Act at once, not three days later. You will sleep better and the patient will also. If necessary, take the case to a nose and throat man and get that root out. If you don't, a maxilliary sinus infection will develop. This is the only way to handle such cases, I have seen many mean cases that have gone on to litigation and other complications, from leaving roots in antrums, packing them open, and reinfecting them over a period of months. I feel, as Dr. Shearer said, that infection ensues in many cases from constant packing. I have opened six antrums in one afternoon in the ordinary extracting of teeth and in no case did maxillary sinusitis develop. I believe that we can open the antrum easily and readily, and have no cause for worry if it is treated promptly afterwards by nasal drainage. The lining membrane, as Dr. Shearer says, does get out of the way in many cases and we do not have to puncture it every time. Many times, it is already punctured or necrotic, but we should avoid it, of course, as much as possible. Replying to the question asked by Dr. Sobell regarding an infected vital pulp—I don't know what I would do. I believe that if a tooth did not respond to a vitality test, I would take the tooth out and have it cultured, if it is anywhere near the antrum. If you are not sure of its vitality, if you think it is going to become pulpless in three or four weeks or a month, take it out. I believe in removing all pulpless teeth that are near the antrum, as no root amputation, root treatment or anything else will serve the purpose.

CONDENSATION OF PORCELAIN FOR JACKET CROWNS*

By ALVER SELBERG, D.D.S., Minneapolis, Minn.

THE world is indebted to Fauchard, Guillimeau and other contemporary dentists of the eighteenth century for the invention and introduction of artificial teeth made from mineral paste or porcelain. The contribution of these men has had a far-reaching effect on dentistry, and in the profession, their names may fitly hold a place of honored remembrance.

Previous to the eighteenth century, ivory and various animal substances were used for this purpose, but all of these were rapidly discarded for the more natural and superior porcelain tooth. From the eighteenth century on, this phase of dentistry has seen many changes, relative to shading, tooth form and structure in general, and, as a substitute for lost or broken down teeth (wherever indicated), nothing to date has taken its place. Accordingly, much is in store for those who seek the highest art in dentistry, a field that is only in its infancy, with unlimited possibilities when we realize how much can be accomplished in bringing about perfection with simplicity.

The primary purpose of any porcelain tooth in the mouth is substitution without detection. As there are no pits and air pockets in the body of or on the surface of the natural tooth, it is reasonable to assume that the closer to complete condensation we arrive in porcelain work, the more natural in appearance will the finished product be. Magnified, the perfect piece of porcelain should present a felted mass of minute crystalline rods imbedded in a more glassy substance. Only by forming some such mental picture, which is in harmony with the knowledge obtained by a microscopic examination of the material, can we understand wherein the real beauty of porcelain resides.

The question of the glaze, or surface finish, holds an extremely important place, for this is the distinctive mark of the finest porcelain and the source of its subtle beauty. In true porcelain, the glaze is composed of materials analogous to those used as the fusible ingredients of the paste and as such materials are refractory, they need to be fired at the same high temperature required to porcelainize the body. Under such circumstances, we can readily understand the place condensation of the body of porcelain plays in producing the velvety tone and deep richness found in the perfect piece.

When light falls on a piece of true porcelain, it penetrates the layers which, so to speak, filter, soften and subdue it, so that the lowest depths shimmer and glisten with the light reflected to the observer's eye through successive envelops of more translucent substance. We can then understand that a porous body of porcelain will, in like manner, reflect

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the air pockets, pits and voids through the glaze to produce a glassy, frosted appearance instead of the smooth, solid finish found in the natural teeth. The voids and flaws in the body of the porcelain when glazed will only be accentuated, and the minute bridges of the glaze over the air pockets fracture to form crypts, which collect residue and appear as black specks in the surface. Therefore, to arrive at the finest in porcelain, the body must be a dense homogenous mass, because, in reality, it enters into and becomes part of the glaze.

Cylinders of equal size, made of high fusing mineral paste, showed a shrinkage of from 13 mm. to 12 mm. in diameter for the condensed porcelain; whereas, the cylinder built up by the ordinary methods shrank to 11 mm. in diameter. There is a shrinkage of approximately twice as much for the uncondensed body. Undoubtedly, then, condensing of the mineral body plays an important rôle, as it aids materially in controlling the shrinkage caused by the fusing. This is a great aid in establishing marginal fits, for both crowns and inlays.

Tests made on baked cubes or porcelain 9 mm. square showed a crushing strength of 65,200 pounds per unit (square inch) for the condensed porcelain, and 55,910 pounds for porcelain made in the ordinary way. This is an increase in strength of nearly 10,000 pounds per unit (square inch). Strength in ceramics is obviously of great value.

The object of this paper is to call the attention of the profession to an existing condition of imperfection in dental ceramics which is obvious to most of us, namely, pits, voids and air pockets found

Fig. 1.—Amalgam die for jacket crown.

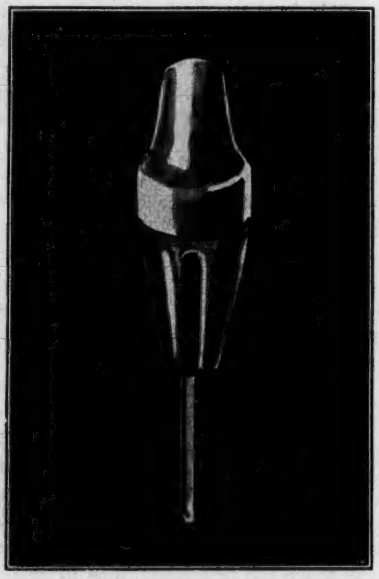

Fig. 2.—Part of porcelain placed on die.

in the body and surfaces of the jacket crowns, as well as in most other porcelain restorations now used in dentistry. It will undoubtedly be conceded that the foundation of the finest porcelain has basic quality in condensation. It is with this thought in mind that this paper is presented, and, for that reason, minor points

Fig. 3.—Celluloid tooth form placed on die.

in the construction of porcelain jacket crowns will not be discussed in detail.

Before starting work, a modeling compound impression should be taken. When flowed up with plaster, this will serve as a study model as well as showing results before and after. The tooth is then prepared for the reception of the crown. A modeling compound impression of the prepared tooth is taken in a copper band. Two plaster impressions of the preparation should include several adjacent teeth. The reason for the second impression will be brought out later. A modeling compound impression of opposing teeth is then taken.

The copper band impression of the prepared tooth is packed with amalgam, and a brad driven into it while still soft. When the amalgam is set, the die is trimmed down, as is generally done, and the head of the brad cut off approximately

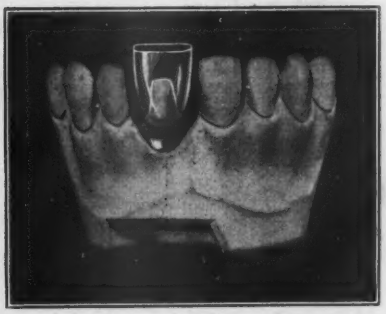

Fig. 4.—Celluloid tooth form on model.

one-fourth inch below the amalgam. (Fig. 1.)

The die is seated in one of the plaster impressions of the case, and flowed up with plaster for the cast. When set, it is separated, trimmed and mounted with the bite for articulation. A platinum matrix is burnished and trimmed so as to have an apron of about 2 mm.

Porcelain selected for the gingival shade is padded on at a 45 degree angle from the gingival margin up to the wall of the preparation. The remainder of the preparation is covered with a thin layer of porcelain applied with a small brush.

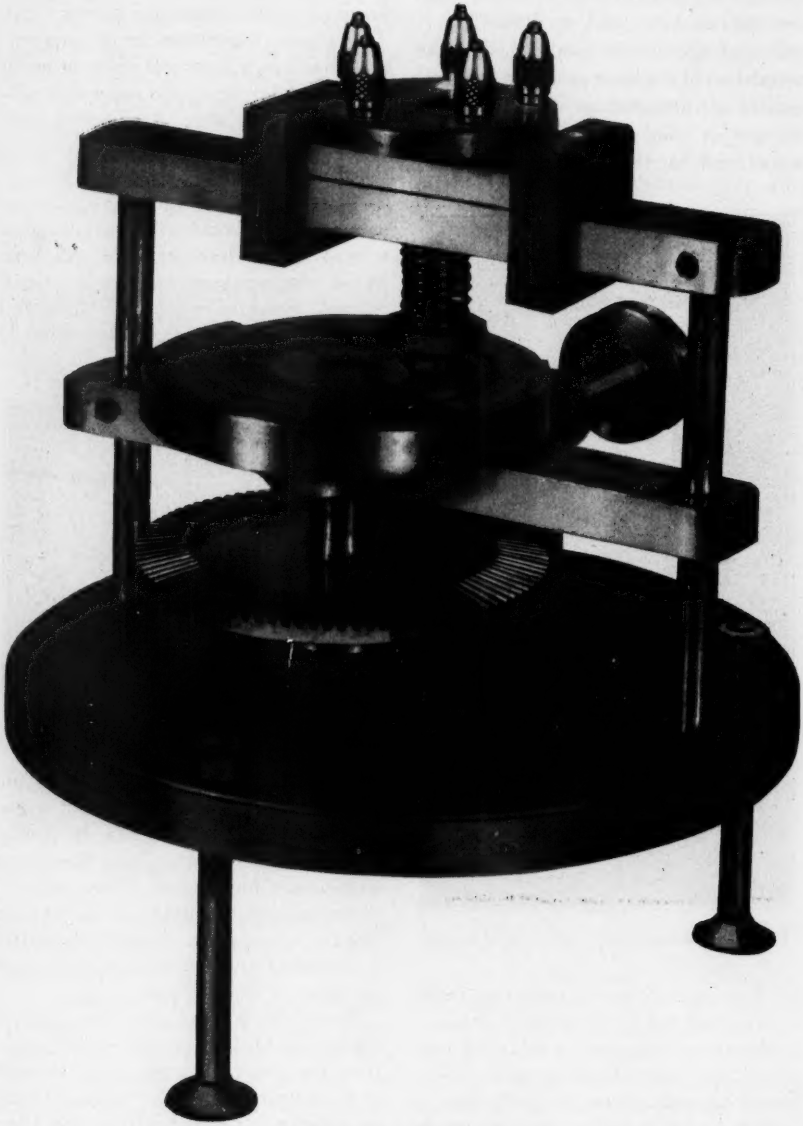

Fig. 5.—Condensing machine.

(Fig. 2.) It is then fired to a high biscuit bake. This then serves to make an air-tight matrix, which is imperative in this method of building a crown, for if there is an escape of air through the matrix, it will tend to form air pockets in the body of the crown. It also keeps the porcelain from flowing between the die and the matrix during its condensation. The collar of the porcelain at the gingival aspect or occlusal aspect of the crown. This provides a reservoir for excess water and for the settling of the porcelain during its condensation. (Fig. 3.)

The die is now used in the second impression of the case, and the plaster cast is made. When this is separated, the contact surfaces of the adjacent teeth are removed approximately 1.5 mm. This second model serves two purposes: first, providing a guide for establishing alinement when the celluloid tooth form is attached on the die; and, secondly, allowing the overbuilding of contact surfaces to compensate for the shrinkage of the porcelain which takes place during the firing. (Fig. 4.)

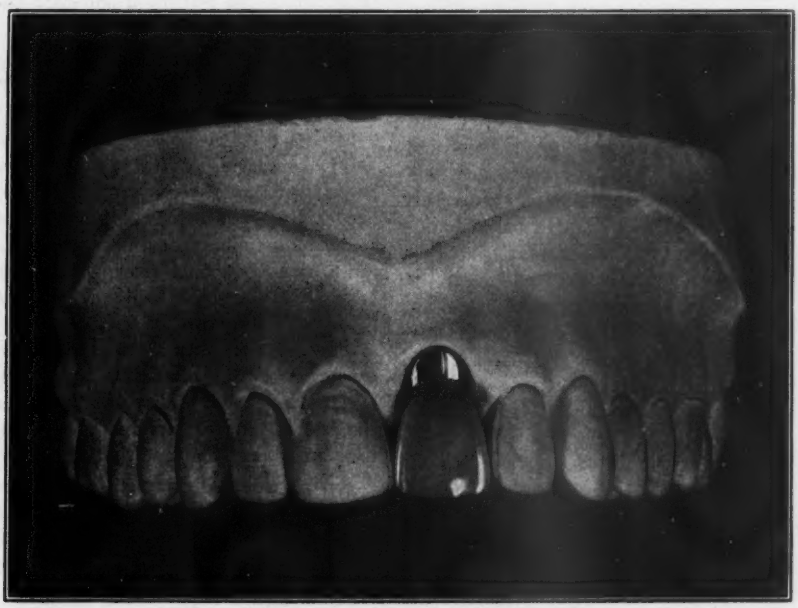

Fig. 6.—Completed crown.

controls the shrinkage and prevents distortion of the matrix.

The matrix, with the biscuit bake, is placed on the die and a collar of sticky wax is run over the apron of the matrix 0.5 mm. from the shoulder. The celluloid tooth form, or tube, is now contoured at the gingival and attached to the sticky wax. This matrix should extend about one-fourth inch over the finished incisal

The die with the celluloid matrix is now placed in the condensing machine, where it is held by the brad which fits

into the pin vise of the condenser.

The machine shown in Figure 5 is run by a motor or lathe, so constructed as to cause a sudden jar when the plunger which holds the die is forced onto a cam having two drop-off steps.

The porcelain is mixed thoroughly on a slab as is usually done. The shades of porcelain corresponding to the teeth to be matched are then placed in the celluloid matrix and blended with a small but long bristle brush. When the matrix is filled, the condensing machine is then started and run at a speed which would cause 200 or 300 drops to appear per minute. The jarring caused by this packer brings the moisture, bubbles and many impurities to the surface, where they are removed by blotting at intervals with a dry towel. The technic of building a crown may be different from that described above, this depending on the shading and blending desired.

When moisture stops rising to the surface, the die is removed from the machine. The celluloid matrix is then slit with a small sharp lance, after which it is removed. The sticky wax on the apron of the matrix is then cut or chipped off, and the die placed back in the cast, which has had the contact surfaces removed. It is then carved to approximate form and is removed from the die and fired to a high biscuit bake, after which it is ground to contact and occlusion in the accurately mounted casts.

When the baking is completed, the platinum apron is trimmed off at the gingival aspect and the crown placed in the mouth, where the finer markings, contact points, etc., are verified. This procedure being completed, the crown is ready for glazing, after which the matrix is removed and the inner surface etched with hydrofluoric acid. Figure 6 shows the completed crown.

The shade of cement used in setting a crown is of extreme importance, for, by using the wrong cement, an otherwise perfect shade of the crown may be altered. For this reason, it is advisable to test the shades by mixing the powder with water and trying the crown in the mouth before the final setting.

CONCLUSION

Realizing an existing condition of imperfection in the texture of porcelain and knowing the importance of texture as a factor in esthetics, strength, and compatibility of the tissues involved, I am submitting this method of obtaining condensation in porcelain jacket crowns.

The greatest crushing strength of porcelain is attained when it has been fired 200 degrees below its fusing point. It has been found that a natural surface finish of a condensed crown can be arrived at at from 20 to 40 degrees less than in the crown made in the ordinary manner. This is then a factor in gaining greater strength in the crown and largely eliminating the possibility of burning out the coloring ingredients and overglazing the porcelain at the time that it is fused.

After glazing is completed, often corrections, such as the grinding of contact points and modification of the length, are necessary. This may be done without reglazing the crown, for a high finish can be obtained by polishing with disks or pumice. This gives a surface as beautiful as the original glaze.

Tests made show a shrinkage approximately twice as great for the uncondensed as for the condensed body of porcelain. It is, therefore, desirable to get condensation, as it aids in securing marginal fittings.

In a natural tooth, there are no pits or airpockets. For this reason, it is obvious that the denser the porcelain, the

better the chance for harmony and blending with adjacent teeth.

From tests made on baked cubes of porcelain 9 mm. square, it was found that condensed porcelain showed a crushing strength of 65,200 pounds per unit (square inch) and 55,910 pounds for porcelain made in the ordinary way, an increase in strength of approximately 10,000 pounds per unit (square inch). Strength is obviously of great importance in ceramics.

It is through this branch of dentistry, which represents the highest art in the profession, that extreme satisfaction can be obtained both by the dentist and those seeking this type of service. It is my hope that this simplified method of condensation will stimulate interest and result in a greater endeavor to attain perfection.

DISCUSSION

A. O. Klaffenbach, Iowa City, Iowa: This method is very similar to the one described by Dr. LeGro, except that he uses a paper instead of a celluloid matrix. The trouble with that method has been in the shaving and blending, especially in the anterior teeth. It is very satisfactory for posterior teeth. There is no question that one can get a very thorough condensation of porcelain. I would like to have Dr. Selberg tell us how he would handle the anterior crown, in order to blend the shades properly.

L. E. Myers, Omaha, Nebr.: Dr. Selberg offers something new, and very different from any other method that we have had in condensation of porcelain. Dr. Selberg arrived at each point definitely and concisely. I question whether it is necessary to go to all of this trouble to get condensation of porcelain. It is true that generally the condensation of porcelain is very poor, leaving it very porous. It seems to me that in our recent practice with ceramics, we are getting much better condensation by sucking out the moisture in the porcelain, than we used to. I believe that the old technic in the condensation of porcelain was to burnish with a spatula. Next came the whipping with a sable hair brush. In our present method of condensation, we use porcelain very moist, almost in a running stage. We apply it to the matrix, and dip it into some dry porcelain powder. This acts the same as the condensation of the sand on Daytona Beach, the race track for automobiles. That is, the suction of the water out of the sand condenses it until we have a perfect race track. The same is true with porcelain. In our office, we use this method, also the sable hair brush. By sucking the moisture out, and then condensing by whipping with the brush and repeating the suction of the moisture, we find that we can get a condensation of the porcelain that makes it very easily handled. In fact, we can drop it on the floor, and practically no porcelain comes off. I may be wrong, in regard to Dr. Selberg's first step of covering the matrix with porcelain. It seems to me that, in order to stop the drawing of that porcelain, one would get a slight drawing of the matrix. I have not tried that. The three-cornered apron, built up to the side of the matrix is unquestionably a good idea. I also agree to bringing the first bake to a high biscuit, which not only shuts out the air pockets, but also minimizes the shrinkage of the porcelain. I gather that Dr. Selberg bakes this in two operations. It seems to me that is a little too quickly. We use three bakes at least in our office.

Dr. Selberg (closing): I usually place shellac on the first baking of porcelain. That allows the shrinkage to take place upward, and I take that into consideration. Regarding the shading for anterior teeth, that can be done by successive bakings, by glazing, with a celluloid matrix, and placing the gingival glaze on and baking it and removing it. Then the incisal color can be placed, and the model made in the same way by placing the sticky wax on the gingival portion and building the celluloid matrix and baking that. For the incisal shades, I cut away to the convexity, and usually the shade runs up to the mesial and distal aspects of the teeth. I then take a wafer of wax, press it against the lingual aspect of the crown and bring it up around the crown on the incisal aspect, and that leaves two folds on the mesial and distal corners. I pinch the wax and foil together and trim it so it is 2 mm. high. Thus a pan is formed which has the wax on the side and the labial surface as a base. I can place the incisal color, hold it in the machine, peel off the wax and bake it.

THE NEWER KNOWLEDGE OF HYGIENE IN DIET*

By J. SIM WALLACE, M.D., D.Sc., L.D.S., F.A.C.D., London, England

IT had seemed to me that I had said enough and been sufficiently explicit with regard to the views on diet which are associated with my name. Repeated requests for another communication seem rather to indicate the need for some further observations. Moreover, I am not at all sure that my views have always been correctly stated by others when they were not in sympathy with them. This at least is my apology for making the following somewhat general review of the subject.

At the beginning of the present century, there was what was claimed to be a science of dietetics based on the caloric value of foods. Physiologists and dietetists had worked it out; and whether you liked it or not, you had to accept it. If you questioned anything, no one took any notice. At best, you were politely told that there was no room in the established science of dietetics for food faddists. If you ventured to base arguments on subjects associated with anthropology and biology, or anatomy and physiology, a deaf ear was turned to you.

If you referred to the biologic principle that animals became adapted to the nature of the environment in which they lived, and contrariwise that the nature of the environment in which the ancestors of man had lived for countless generations gave a clue to the nature of the environment for which he was adapted, you were told, "We don't want theory." If you tried to contend that the hygiene of the mouth and alimentary canal might be overlooked when attention was concentrated on nutrition exclusively, your plea was liable to be treated with indifference on the grounds that the hygiene of the alimentary canal had nothing to do with dietetics, or if it had, the toothbrush and an antiseptic mouthwash would keep the mouth and teeth in a hygienic condition, and the alimentary canal could look after itself. If you tried to indicate the incongruity of advocating a pappy milk diet for children after they had a first class complete set of teeth, as excellently adapted for dealing with solid food as, and often more excellently than, were the teeth of adults, you might have been told, as I was, that the science of dietetics was for physiologists and medical men, and that I had better devote my attention to the teeth. I had indeed devoted my attention to the teeth, but, here again, although I brought knowledge of a biologic nature to bear on the subject, and indicated that the theories of hereditary degeneracy of the teeth and many of the ridiculous ideas then prevalent with regard to the cause of caries, irregularities and pyorrhea were erroneous, it was noted that my views had "been received with the studied indifference which is characteristic of the attitude of the more responsible members of the dental profes-

*Read before the Dental Officers Group of the Society of Medical Officers of Health, Feb. 27, 1931.

sion in England toward original observation and research."[1]

In order to get a little closer to my subject, perhaps you will allow me to indicate why I came to take an interest in dietetics. In the first place, I became somewhat fascinated with the principles of biology in general, and questions associated with heredity in particular. Then, becoming a dental surgeon, I began to take an interest in teeth, and the various theories in vogue at the time with regard to the causation of caries and the irregularities of the teeth. As a result of these interests, it became increasingly difficult to accept current ideas with regard to the supposed hereditary degeneracy of the teeth and the prevalence of caries. Daily observation and a knowledge of the chemicoparasitic process led me to think that the trouble originated in changes in the food which rendered it liable to lodge and ferment in certain situations about the teeth.[2] I recognized the valuable work that physiologists had done in the field of nutrition and argued that since the nutritive qualities of our food were as good as, if not better than, those of almost any other animal, it was unreasonable to think that there was any likelihood of defective nutritive qualities being the cause of the prevalence of dental disease.

I cannot but presume that you are all to some extent familiar with my views, whether you recognize them as mine or not, for, as the *The Lancet* pointed out, they are often brought forward "without sufficient acknowledgment to their author."[3] I intend to recall some points and indicate how far they have progressed and also how far they have been ignored. To do this, I must ask your indulgence in letting me refer to some of my earlier books. Commenting on my first, on the cause and prevention of dental caries, written now over thirty years ago, the *British Dental Journal* said, "If not absolutely convincing, is none the less of great importance in calling attention to the dental dangers arising from our civilized methods of preparing food."[4] Perhaps I may say most of the criticisms on this book at the time seemed quite satisfactory to me. Nevertheless, apart from a discussion in *The Lancet,* for several years afterwards but little attention was given to the subject in either medical or dental literature. Indeed, as already indicated, it was noted that the responsible leaders of the dental profession, with but few exceptions, rather ignored the whole subject, and but little prescience was shown with regard to the now recognized "widespread influence"[5] which they apparently have had on medical literature and the science of dietetics generally. I persisted on the same lines, and before half a dozen years were over, had written some "Supplementary Essays on the Cause and Prevention of Dental Caries," together with a little book on "The Rôle of Modern Dietetics in the Causation of Disease." In the former, I tried to draw attention to some physiologic processes and attributes of the saliva with regard to the self-cleansing or hygiene of the mouth. In the latter, I tried to indicate that our methods of selection and preparation of foods were dangerous not only from the point of view of oral hygiene, but also from the point of view of the whole alimentary tract. Commenting on

1. T. E. Constant: Tr. Odontol. Soc. Great Britain, March, 1903, p. 143.

2. Wallace, J. S.: Cause and Prevention of Decay in Teeth, 1900.

3. Lancet, Jan. 5, 1924, p. 28.

4. Review, Brit. Dent. J., 1900, p. 538.

5. Matheson, Leonard, and Payne, Lewin: History of Dentistry in Great Britain, Proc. Seventh Internat. Dent. Cong. p. 2213; J. A. D. A., 15:441 (March) 1928.

my book on modern dietetics (1905), the *British Medical Journal,* admitting my contentions, said, "We agree that there should be a return to a more natural plan in the feeding of children." Nevertheless, for several years afterwards, little attempt was made to advocate any rectification of the errors in diet to which attention had been drawn, and the calory dietetists still ignored certain accessory food factors whose influence and importance it seemed to me had to be taken into account. It was not until my book on "The Prevention of Dental Caries" was published in 1911 that something like a full recognition of the truth of the principles advocated was made and the whole system seemed destined to supersede, or rather improve on, the dietetic system of the past. One of the leading articles on the subject indeed said, "In the last few years the diet of children has been entirely reconsidered. Much of the traditional feeding has been rejected, and in its place a carefully thought out dietary based on scientific principles has been established."[6] Though my views appear gradually to have become accepted by the dental profession also of this country, there was at times, no doubt, some reluctance to accept everything in its entirety, but, up to 1914, things were progressing in a fairly satisfactory way. When the war came, the ensuing consternation diverted attention from questions associated with dental caries. Concern naturally was concentrated on how to get food at all, and to have this properly rationed. Nevertheless, certain events during the war had a special relation to diet and dental caries, and threw some light on the subject. Perhaps, the most important point was the relative scarcity and increased price of sugar. Other points of importance were no doubt the scarcity of and consequent decrease in the consumption of milk, the production of rather coarser bread and the fact that the crusts were eaten instead of being cut off and thrown away as in prewar times. These facts all theoretically tended to reduce the amount of caries, and subsequent work revealed the fact that there had been a marked reduction in the prevalence of caries among children. After the war, the price of sugar fell, the tax on it was reduced and its consumption began to rise again. Unfortunately, too, since about the time that the consumption of sugar increased, there has been an increased advocacy regarding consumption and increased consumption of cod-liver oil, together with malt extract or other sugary mixtures, and this is now being reflected in statistical inquiries with regard to the incidence of caries. So far, apparently, it is the preschool child that has suffered. It is during the preschool period that young children are most liable to develop rickets. To avoid this possible contingency, cod-liver oil and malt is widely administered, and so it is the children at the "age-5 period" who reflect the consequences of this misguided care. Sir George Newman refers to a statistical inquiry in the Cambridge Borough Report which indicates the percentage of carious temporary teeth in the 5-year-old group of children in the borough from 1909 to 1929 inclusive. It states: "Between 1917 and 1922 this percentage fell steadily from 35 to 12, while from 1922 onward it has been rising again and now stands at 21."[7] Another fact which presumably accounted for the general fall in the incidence of caries was appreciation of the nature of the cause, and that it was a preventable disease. Something in the nature of a campaign to prevent caries

6. Wallace, J. S.: Diet in Childhood, Univ. Med. Rec., May, 1912, p. 412.

7. Health of the School Child (for 1929), 1930, p. 56.

was carried on by the late James Wheatly, and leaflets designed to bring home even to the ordinary layman the salient features of the new régime were widely distributed in his county. Some medical men and not a few dentists, recognizing that the older toothbrush and antiseptic mouthwash method could not be relied on, tried the system, and, as time went on, were able to report excellent results. To cut a long story short, the newer ideas were successful, and lavish praise, whether merited or not, was accorded the system. This did not go unnoticed by dietetists and research workers, and interest began to be taken in diet from a point of view other than that of nutrition.

Although, in the year 1881, it had been shown "that it is not possible to keep animals in health if they are fed on the artificially purified constituents of food, proteins, fats, carbohydrates, salts and water,"[8] for about twenty-five years, little or no special notice was taken of this fact. In the latter half of the first decade of this century Sir Gowland Hopkins began to take an interest in the subject, and later was able to show that "the addition of a small quantity of fresh and natural food, such as milk, sufficed to maintain the animals in good health."[8] To the unknown substances in milk which restored the animals to health, he gave the name "accessory food factors," and suggested that they were of a stimulating or catalytic nature. The accessory food factors to which he referred were not, or at least not the only, accessory food factors to which I had tried to draw attention. I referred more particularly to the physical nature of the food, together with its qualities resulting from aromatic and acid as well as stimulating principles, and concerned myself more especially with the hygiene of the mouth.

8. Harmsworth's Home Doctor, p. 4282.

Following on the introduction by Funk of the attractive but intoxicating and misleading catch-word "vitamin," and particularly during the last decade, we have been overwhelmed with research and discussions which seem at least to justify my early attempts to point out the dangers resulting from modern methods of selecting and preparing foods. In fact, these dietetic dangers are now recognized, and a return to a more natural and rational system of dietetics is widely advocated.

Now let me remind you of the food factors which I considered valuable from the point of view of oral hygiene. First, let me refer to such physical properties of the foods as tended to stimulate mastication. The importance of these was indicated with regard to the removal of bacterial plaques and polishing the teeth, the prevention of irregularities, the prevention of tartar formation, the prevention of recession of the gums and later[9] from the point of view of the peristalsis and hygiene of the alimentary canal. I repeatedly used the words "coarse" and "fibrous" to indicate the physical nature of the food claimed to be desirable, and indicated that its consistency depended largely on the cellulose that it contained. Though my meaning remains the same, I now prefer to speak of "crisp" and "fibrillar." The words "coarse" and "fibrous" are unattractive, and besides do not distinguish the desirable physical characteristics so well. Modern dietetists seem really to refer to exactly the same thing when they speak of "roughage," and their experiments on monkeys which show the beneficent effect of filter paper, that is, cellulose, in alimentary hygiene, indicate that we were not far off in our claims.

9. Wallace, J. S.: Rôle of Modern Dietetics in Causation of Disease, 1905.

Next, let me refer to the stimulating principles. In the course of my investigations, it happened that I wanted some saliva for experimental purposes. I had read that this could be got by chewing a toothpick. I tried this method, but found that the amount secreted was rather scanty and secretion was slow, whereas a small amount of acid or sugar called forth a relatively rapid and copious flow. From this, it was obvious that although masticating an inert substance such as a toothpick did stimulate the secretion of saliva, it was not nearly so effective as certain soluble substances which no doubt acted reflexly through the taste organs in the tongue, and possibly, in the case of aromatic substances, reflexly through the olfactory organ. As already indicated, Sir Gowland Hopkins suggested that certain "accessory food factors" had a stimulating or catalytic influence. Later, Dr. Cramer, who was acquainted with my views, in conjunction with Dr. Kingsbury, directed attention to the hygiene of the alimentary canal, and showed that when animals are deprived of vitamin A, "the mucous glands atrophy"[10] and the alimentary canal becomes unhealthy. To be more explicit, they said that "in a normal animal the intestinal bacteria are restricted to the central lumen of the intestine," but lack of vitamin A deranges the alimentary canal; "the intestinal bacteria penetrate between the villi into the crypts of Lieberkühn and into the mucous glands of the caecum, which they fill and where they proliferate." It would appear that an absence of vitamin B gives rise to a similar atrophy of the lymphoid tissue. Now, as it seems to me, it is just the mucous glands and the lymphoid tissue which are of special importance in alimentary hygiene. As noted elsewhere,[11] mucous provides "a suitable medium in which phagocytic corpuscles may perform their scavenging work. Thus, while lubricating the mucous membrane, it also appears to help indirectly to keep the surface free from obnoxious micro-organisms, or at least to restrain them from getting a foothold where there is danger of this taking place." Thus, the important point has been noted that when food is deprived of its stimulating principle, the hygiene of the alimentary canal is not properly provided for. Unfortunately, being unable to emancipate themselves from the old ideas of nutrition, but few other research workers have recognized that the hygienic value of accessory food factors is of great importance, and volumes of talk about the newer knowledge of nutrition has obscured the value of accessory food factors as regards the hygiene of the mouth and alimentary canal. Of course, as far as we are concerned, this is probably only of academic interest, as the amount of vitamin A required is so "infinitely little" that, in civilized communities, except under peculiar and extreme maldistribution or selection of food, no disease or harm is likely to result. Indeed, it would seem that we in civilized countries are probably better supplied with vitamin A than are many savages and animals.

With regard to acids in the food, these too, of course, under the calory system, had no nutritive value, but as substances of hygienic value, food acids have considerable importance, not only from the point of view of calling forth a liberal supply of alkaline saliva and so correspondingly being of value in the physio-

10. Cramer, W., and Kingsbury, A. N.: Local and General Defences Against Infections and Effect of Them on Vitamin Deficiency, Brit. J. Exper. Path., 5:302 (Oct.) 1924.

11. Wallace, J. S.: Physiology of Oral Hygiene, Ed. 2, London: Balliere, Tindall and Cox, pp. 223 and 224.

logic means of removing carbohydrates from the mouth or rendering acids harmless, but also on account of the share they take in preventing the formation of tartar, which so generally leads ultimately to pyorrhea.[12] This aspect of the subject, important though we know it to be, has received but little attention outside the confines of the dental profession, as the physiology of oral hygiene has been almost completely neglected by physiologists. Though a few medical men now recognize that the primary function of the saliva and oral mucus is to keep the mouth in a hygienic state, physiologists still seem to be oblivious of the fact. Thus, Drs. Lyle and De Souza in their otherwise no doubt excellent "Manual of Physiology," under the heading "digestion," say, among other things, that "the functions of the saliva are . . . to stick the portions of masticated food together and consequently to form the bolus. The bolus of food is lubricated with mucin and so prepared for being swallowed. . . . by the action of its amyloclastic enzyme ptyalin, to convert cooked starch into maltose.[13] It would thus appear that they are quite oblivious of the function of the mucus and saliva from the point of view of oral hygiene, erroneous in their ideas about the formation of a bolus and unconcerned about the fact that man is the only animal who normally eats cooked starch, though all land animals are provided with salivary glands. If any part of my earlier work has given rise to more unanimous approval and less adverse criticism than another, it is with regard to the physiology of oral hygiene. It is rather a serious matter that physiologists have failed to appreciate what others now regard as an elementary truth, namely, that the mouth of man and animals is kept in a hygienic state by physiologic processes when the food is of such nature that normal processes are not stultified or hindered from being effective. D. D. Rosewarne, in his book on "The Science of Nutrition, Simplified," refers to the fact that "the new view of the function of the saliva has not found its way into the textbooks." (p. 298.) But until the medical profession, including budding medical officers of health, are taught these physiologic processes and appreciate their value, substantial progress will be slow and precarious. Surely, the matter is urgent. It has repeatedly been stated, and no physician, dentist or medical officer will contradict it, that the unhygienic state of the mouth of men, women and children is a disgrace to civilization, yet physiologists are as mute as if all were well and there was no need to say anything more on the subject. I do not say all physiologists, because occasionally one does come across statements from physiologists which indicate appreciation of the new views. Thus, in his story of digestion, Bainbridge says:

> Saliva also plays an important part in dissolving food materials and so initiating or increasing taste sensations; it makes speech possible by keeping the tongue and lips moist; and, last but not least, it cleans the mouth at the end of a meal. Hard, raw fruit, it may be added, is the ideal last course of any meal. It not only helps to clean the teeth from particles of food which, if allowed to remain between the teeth, might decompose and help to cause dental decay, but it also helps to stimulate a further flow of saliva—the natural cleanser.[14]

12. Wallace, J. S.: Teeth and Health, London: Faber and Gwyer, Ltd., 1926, Chap. VII, p. 156.

13. Lyle, H. W., and De Souza, David: Manual of Physiology for Students and Practitioners, Ed. 3, New York: Oxford University Press, 1930, p. 94.

14. Bainbridge, J. S.: Human Machine, Chap. VIII, New Health, March, 1930.

I sometimes think too little attention is paid to a certain form of malhygiene which eventuates in pyorrhea, and, for this reason, I should like to say a word on this subject. In my earlier books, I noted the value of fibrous foods such as salads and fresh fruit in the prevention, not only of caries but also of the irregularities of the teeth and of tartar and recession of the gums. At first my hypothesis was that if we keep to foods in their natural state, especially to those requiring mastication, we should not be likely to be troubled with these diseases. Observation and knowledge derived from physiologists, more particularly the work done on mucus by Prof. W. J. Gies and his collaborators, led me further to appreciate the value of the natural acids which were contained in these foods from the point of view of the prevention of tartar, and, to cut a long story short, indicated that the foods most useful for keeping tartar from becoming deposited were, "those which necessitated mastication—salads and fresh fruits at the end of meals" being "particularly helpful."[15] It has been claimed that there is a striking resemblance between the detergent diet which I recommended and that now recommended by the advocates of fruit and vegetables on account of their containing vitamins, which are supposed in some mysterious way to be "protective" foods. As far as the teeth and gums are concerned, it is unnecessary to assume anything mysterious, for the explanation given of the detergent effects from a mechanical and chemicophysiologic point of view indicates quite clearly that it is friction and acidity of the food and the qualities of the mucus and saliva stimulated and secreted which keep the teeth and gums free from objectionable deposits. Mastication is not stimulated by mysterious substances, it is the physical nature of the food which does this, and if teeth are not used through absence of opposing teeth or for other reasons, tartar tends to be deposited on them, while other teeth that are well used are kept free from deposit. It is thus obvious that the unhygienic state of the mouth associated with the deposit of tartar is local, and depends largely on malhygiene and lack of function, and that it is not likely to be due to constitutional conditions except when these diminish functional activity or have some specific action on the gums; nor is it due to the absence of vitamins, except that these may have a stimulating effect on the glands of oral hygiene. Vitamins are amply provided for in the detergent diet which we advocate.

It has also been noted that the resemblance in the dietaries advocated by the vitamin-diet and the hygienic or detergent-diet schools of thought breaks down completely when we consider cow's milk as an article of diet after the weaning period. As has long been known, from the point of view of nutrition, cow's milk contains everything that is wanted — proteins, fats, carbohydrates, phosphates, calcium—and that in most excellent form. It is all no doubt of the highest biologic value. As it is intended for an animal that grows five or ten times the rate of a child, we may rest assured that it possesses all the factors required for promoting growth in full, perhaps excessive amounts. It contains all the vitamins, but ideal food though milk is often claimed to be, it contains remarkably little vitamin D. In fact, it appears to be absent altogether sometimes. Incidentally, it might be noted also that this vitamin is associated with the cream, which, in civilized countries,

15. Footnote 8, p, 2053.

is generally filched by human beings, and consumed in the form of butter. Deprived of vitamin D in this way, the milk is given to calves, but it is children who suffer with rickets, and the men, women and children who suffer from dental caries, while the calf never suffers from the latter disease at least. It might further be noted that it is often overfed children that suffer from rickets, when rachitogenetic additions are made to their dietaries. But that is not the question here. What we want to know is whether milk is conducive to oral hygiene, and as oral hygiene tends to be jeopardized by irregular and crowded teeth, whether milk is conducive to that sturdy development of the jaws which was a marked characteristic of our ancestors before men kept cattle. Dental surgeons have noted that the milk-fed child, though generally perhaps in first-class health, frequently has its teeth crowded and ruined with caries, and its mouth in an altogether unsatisfactory state. I have elsewhere[16] said enough on this subject, and would here only repeat that milk contains all the vitamins, but it certainly is not protective against dental caries. In fact, next to concentrated sugars in various forms—sweets, jam, marmalade, honey, golden syrup, treacle — milk is the food most nearly calculated to destroy the teeth, more especially when given as a sloppy diet or taken with biscuits between meals, or before going to bed. It was a recognition of these facts which no doubt induced the committee appointed to draw up the leaflet on the prevention of decay of teeth authorized by the Society of Medical Officers of Health, to say, "Milk is a food, and should only be taken at meal times, and must always be followed by a tooth cleansing food." I was glad to note in a recent issue of *Public Health* the suggestion made by G. C. M. M'Gonigle that a diet should "be devised which would avoid the absurdity of giving milk after the natural source was no longer present."[17]

The attempt to show that rickets was a deficiency disease has tended to cause much confusion of thought. The subject has occupied medical literature in recent years so fully, and the supposed similarity of the etiology of rickets and caries been so widely canvassed, that some comments on our knowledge with regard to the etiology of rickets seemed to be desirable. The recognition of the value of cod-liver oil in the treatment of rickets seems first to have been made at the beginning of the last century in Holland. It then soon spread to Germany, France and other countries. About the middle of last century, Francis Harris recognized its value for dogs, and when "his pups became rickety from being fed too much on biscuit, he cured them by the administration of cod-liver oil."[18] When I was a student of medicine about forty years ago, we were taught that "careful attention to diet and general hygienic management claim the foremost place both as preventive and as curative treatment,"[19] and that "starchy food should either be suppressed altogether, or restricted in quantity, according to the age of the child and the special requirements of the case; and may be replaced by light nitrogenous food (milk, yolk of egg, etc.)."[20] Medicinally, cod-liver oil was the recognized standby.

16. Wallace, J. S.: Footnote 9; Teeth and Health (Footnote 12); Variations in Form of Jaws, St. Louis: C. V. Mosby Co., 1926.

17. M'Gonigle, G. C. M.: Disc. Burns, David: Pub. Health, 44:111 (Jan.) 1931.

18. Obituary Notice, Brit. M. J., Oct. 3, 1885.

19. Carter, A. H.: Elements of Practical Medicine, Ed. 5, 1888, p. 87.

20. Footnote 19, p. 88.

Since then, new light has been thrown on the subject. In 1889, Huntly brought forward evidence which strongly supported his idea that absence of sunlight was the chief cause of rickets.[21] T. A. Palm, in 1890, also showed the importance of sunlight, and indicated by "the geographical method that rickets abounds where light is lacking and is unknown where light abounds."[22] Soon after the discovery of vitamins, it was assumed that rickets belonged to the class of vitamin-deficiency diseases. "Mellanby suggested fat soluble A, or one closely associated with this factor. When this was shown to be untenable, an unknown vitamin (X) was accused by Park but acquitted."[23] Later, "in Berlin, Professor Huldschinsky showed that rickets could be cured by irradiation with ultraviolet light."[22] Then, Alfred F. Hess, of Columbia University, New York, showed that "rats fed on a rachitic diet recovered if to it there were added portions of the skin of an animal irradiated before death, whereas the addition to the dietary of a skin of a non-irradiated animal was useless. In short, irradiation produced "vitamin D" in the skin. From this, it was a short step, taken notably by Professor Steenbock in Wisconsin, to produce vitamin D by irradiation in foodstuffs."[22] Though not depreciating the value of cod-liver oil, Findlay and Paton stressed the value of exercise, fresh air and hygienic environment and showed that the fat soluble vitamins were not necessary if the diet were otherwise reasonable.[24] Perhaps by way of mitigation of the erroneous ideas of Professor Mellanby, and to try to keep to some vitamin theory, the substance which was produced by irradiation was called vitamin D.[22] But according to experts and as unequivocally stated by Mrs. Mellanby, "vitamins have one important property in common in that only vegetable life can synthesize them."[25] Thus, in their conception of the nature of vitamins, the antirachitic factor, irradiated ergosterol, is not a vitamin at all. Unfortunately, although he has had to abandon his fat soluble A deficiency vitamin theory of the causation of rickets, Professor Mellanby apparently rather misled the medical profession in this country for years, and hindered the appreciation of the newer knowledge of the causation and prevention of rickets, dental caries and the irregularities of the teeth, which from the point of view of both children and adults in civilized communities is of outstanding importance. Nevertheless, he has not been altogether successful, and, even with regard to rickets, there is still certainly no clear evidence that it is a deficiency disease, although its prevalence can be diminished or possibly the disease might be prevented altogether by the substance (irradiated ergosterol) which acts antagonistically to the incidence of the disease. Indeed, it has not by any means been proved that rickets is not caused by a positive substance resulting from unhygienic environment and the administration of unsuitable foods during rapid development.

21. Huntly, William: Habits and Diet of Natives of Rajputana with Reference to Etiology and Treatment of Rickets, Rajputana Press, 1889.

22. Saleeby, C. W.: New Health, October, 1930.

23. Rosenau, M. J.: Preventive Medicine and Hygiene, Ed. 5, p. 633.

24. Findlay, Leonard: Underlying Cause in Pathogenesis of Rickets, J. A. M. A., 83: 1473 (Nov. 8) 1924.

25. Mellanby, May: Experimental Evidence Demonstrating Influence of Special Dietetic Factor on Development of Teeth and Jaws, Dent. Rec., 40:65 (Feb. 2) 1920.

I have read that E. V. McCollum says, "Since rickets is a disturbance of calcium and phosphorus metabolism, disturbances of digestion which interfere with the assimilation of these elements undoubtedly contribute to the development of this disease in children." Again, we may note that it is alimentary malhygiene and not a food deficiency which is probably primarily the positive cause of this disease. Unfortunately, the erroneous ideas of Professor Mellanby with regard to rickets were duplicated by the similar, if not identical, views of Mrs. Mellanby with regard to hypoplasia and "structural defects" in teeth which are not by any means always regarded as hypoplastic, and her views with regard to the etiology of dental caries and the irregularities of the teeth for years misled the medical profession almost *en bloc*. It is recognized that to prevent or cure rickets caused by a rachitogenetic diet or an unhygienic environment, irradiated ergosterol may be required, but apparently this does not apply to teeth, at least to the enamel. Bauer found that "the ameloblasts (or enamel-forming cells) by means of which enamel receives its lime, have quite an unusual avidity for this element, and even in rickets these cells collect so much lime that a normal calcification takes place."[26] Bauer's findings are corroborated by those of Maxwell Karshan who made his research on rats, whose incisor teeth are of continuous growth, the correlation of their development and of the bones being much more easily ascertainable than in the case of dogs. He found that, on a rachitic diet, the amount of calcium and phosphorus in the bone was reduced almost by half, while for the teeth on either the rachitic or nonrachitic diet the calcification was the same. The averages and average deviations of calcium and phosphorus of the teeth and bones are given in the accompanying table.[27] The figures represent the percentage of the dry weight of the alcohol ether extracted material.

DEVIATIONS OF CALCIUM AND PHOSPHORUS OF TEETH AND BONES

	Teeth		Bone	
Diet	Calcium Per Cent	Phosphorus Per Cent	Calcium Per Cent	Phosphorus Per Cent
Rachitic	26.7±0.6	13.8±0.5	11.3±1.2	5.9±0.8
Rachitic + KH_2PO_4 + Cod Liver Oil	26.5±0.5	14.0±0.1	21.8±0.7	10.5±0.5

Only when this "severe rachitic diet" is deprived of calcium carbonate are the incisor teeth affected, and even then the coronal enamel of the incisors and the molar enamel is apparently normal.[28] More by good luck than by design, and perhaps unconscious of the fact, Mrs. Mellanby has shown most conclusively that even though puppies have been subjected to deficiency diets, and vitamin D has been excluded during development, even including prenatal life, and though this has no doubt been done with scien-

26. Bauer, Wilhelm: Etiology of Hypoplasia in Rickets, Vierteljahrsschr. f. Zahnheilk., No. 1, 1929; abstr. Brit. Dent. J., 50: 782 (July 1) 1929.

27. Karshan, Maxwell: Proc. Soc. Exper. Biol. & Med., 27:200-202 (Dec.) 1929.

28. Karshan, Maxwell: Calcification of Teeth and Bones on Rachitic and Nonrachitic Diets, J. Dent. Res., 11:64 (Feb.) 1931.

tific rigor in laboratories under her supervision, as an almost invariable, if not invariable, rule, the teeth of these experimental animals never become carious. I understand that dogs in this country are practically if not entirely free from caries. I gather that vitamin D is rather limited in its distribution. It is found abundantly in cod-liver oil. It is present in the yolk of eggs. Cream and butter contain small but generally appreciable amounts. Nevertheless, children who do get these foods are extraordinarily subject to caries, while dogs, which seldom get these luxuries, manage to keep free from the disease. Mrs. Mellanby tells us that "as a matter of fact, the diet and general metabolism of the domestic dog is as near that of the human being as any other living thing."[25] It seems to follow, if we are to put any value whatever on animal experiments, that deficiency diets, at least diets deficient in vitamins A and D, never cause caries in human beings. It is curious that the obvious corollaries of her experiments should still frequently be overlooked. No doubt, we shall soon have research purporting to show that the absence of these vitamins causes or predisposes to caries in human beings, and, if so, what has to become of the value of animal experiments which show precisely that this does not take place?

I wonder what ideas a little quiet reflection will arouse when the blinding enthusiasm for vitamins has passed? On the one side will be seen the solid tangible work of the dental profession, notably the common sense observations of Parmly and Robertson, carried forward by the work of bacteriologists from Pasteur onward. They will note the reliability of the work of Miller and others who clearly elucidated the nature and pathology of dental caries. They will see the importance of the work of Magitot and Miller in demonstrating the experimental method of the production of the disease. They will observe the outstanding work of Black, Tomes, J. Leon Williams and others, all of whom, notwithstanding some mistaken ideas, contributed valuable and reliable data which helped to lead to the possibility of more or less complete elucidation of the cause of caries, and allowed us to define this clearly and introduce the methods of prevention now widely recognized to be effective. On the other hand, they will note how the medical and dental professions were distracted by vague ideas about metabolism even in inorganic matter. How they were frenzied with hope that the prenatal and postnatal administration of drugs containing vitamins would allow us to dispense with the necessity of attending to the dictates of the physiology of oral hygiene, and alimentary hygiene in later life, and how they were misled by animal experiments which, on analysis, proved little or nothing except that the leaders of thought of the dental profession were right from the first and all the time. The pity of it all is that the knowledge which had been slowly gained by many years of painstaking investigation was obscured for well nigh a decade from those who otherwise should have been disseminating the knowledge which was and still is so urgently required. As the editor of the *Medical Press and Circular* recently said, "A vast amount of disease results from neglecting to apply our present knowledge. . . . We know how dental caries can be prevented. . . . It is exasperating to know that we have it in our power to check an untold amount of disease by the simplest possible means and that we fail to use that power."[29]

29. M. Press & Circ., Oct. 22, 1930.

Nevertheless, we can be optimistic. The vitamin fog is lifting and rays of light are coming through. Indeed, everything seems to have been drifting in the right direction, as is easily seen if we take the trouble to look into matters carefully. It will, I think, be admitted by those who know the literature of the subject that, to all intents and purposes, the theory of the hereditary nature or any inherent constitutional cause of caries, so generally held at the beginning of the century, is now more or less completely abandoned. Indeed, there is almost need now to mention that hereditary predisposition to caries does to some extent exist, as, for example, on account of variations in the form and depths of the carbohydrate retention areas of the teeth. Again, that caries is a disease of dietetic origin is now almost universally recognized. Similarly, apart from matters associated with teeth, the dietetic dangers of our civilized modes of preparing foods are equally generally recognized. That oral hygiene may be maintained by dietetic methods is widely recognized also. Dietetic experts do not now sniff at foods such as fruit and vegetables because they are not sufficiently nutritious. In fact, in general, what we claimed a quarter of a century ago is now recognized as true, and certain aids to the practical elucidation of the subject have arisen. One is that it is now possible to get the authorities in institutions to frame dietaries which allow the substitution of fruit for sugar.

As you know, many tests on individual children have shown that caries is a preventable disease, and as the application of the rules of prevention has given us such excellent results, it has come to be recognized that the methods should be tried in institutions with numbers of children. Tests of this kind have been made, and although so far perhaps none of them are altogether satisfactory, taken together, they show, I think, very clearly what is true and what is not. I have already referred to an investigation of this nature by Mellanby, Pattison and Proud,[30] which seemed to reveal a close association between dental caries and the amount and the quality of fermentable carbohydrates.[31] According to *Science Progress,* this investigation "verified with some statistical exactness the well known hypothesis that caries in teeth is due to carbohydrates."[32] Another similar investigation carried out a few years later gave rise to a long, "lively" and informative discussion in the *British Medical Journal,*[33] the net result of which was to show that the incidence of dental caries was dominated by the amount of sugar in one form or another in children's diet, and that irradiated ergosterol had no influence on it at all. Still another experimental study recently just referred to would seem once again to indicate that treacle is just as detrimental to the teeth as any other form of sugar, and the criticisms of Harry Campbell[34] would indicate that the presence or absence of vitamins A and D has little or nothing to do with the subject at all.

In the experiments here referred to, the teeth that did decay were formed before any vitamin diet was designedly given. So that, presumably, "defective structure" was equal both in the group

30. Mellanby, May: Pattison, C. L., and Proud, J. W.: Effect of Diet on the Development and Extension of Caries in the Teeth of Children, Brit. M. J. 2:354 (Aug. 30) 1924.

31. Wallace, J. S.: Diet and Dental Caries, Brit. M. J., 2:486 (Sept. 30) 1924. Footnote 12, pp. 144-147.

32. Sc. Prog., 19:495, 1924-1925.

33. Brit. M. J., Dec. 22, 1928; Feb. 2, 1929; April 13, 1929.

34. Lancet, December, 1930.

in which the incidence of caries was high and in that in which it was low. If vitamin D had anything to do with the incidence in the different groups, it surely must have been via the saliva. In others words, it was made clear that the welfare of the teeth is dependent on oral hygiene or hygienic conditions external to the tooth, rather than on nutrition. In America, experiments with large groups of children have been carried out by R. W. Bunting and his collaborators. They state that "by experimental feeding of a well balanced diet from which sugar and candy were practically eliminated, to a large group of children over a period of one year, it was found that they developed normally and were in good health, and that the growth of *B. acidophilus* in the mouth and dental caries were markedly reduced."[35]

Evidence is sometimes best when no preconceived theories have influenced the carrying out of the experiments, and, for this and other reasons, we may refer to the observations of Mr. S. Dunn, senior dentist, Glasgow education authority, in institutional schools where he found "dental decay, tartar and infected gums prevalent in children of those institutions where the dietary is rich in soft starchy foods and deficient in the more cleansing fibrous varieties." But he says:

> In visiting the Langside School for the Deaf for a number of years, I cannot recollect any mouths in the condition just described. I have made inquiries into the routine of diet, and found that the clean mouths were wholly attributable to the Sim Wallace doctrines being unconsciously carried out—a good quantity of fibrous food requiring at dinner a knife and fork, and fresh fruit daily at the end of each meal. I find at other places, even when meat is given, it is minced, and all partaken of with a spoon; milk puddings a frequent item in the menu, but no fruit to follow except rarely. The difference between two institutions like these is so striking as to be worthy of note.[36]

Vitamin enthusiast though he may be, Corry Mann brings forward complimentary evidence of a different but equally satisfactory nature. He referred to the fact that "in Sim Wallace's opinion the principal cause of dental caries was deficient cleansing of the teeth in association with sugar lodging about the crevices and retention areas of the teeth." This view had received support from observations which he (Dr. Mann) had made a few years ago during dietetic investigations on boys of school age living in an institution near London. Though the basic diet was low in caloric value and only 18 per cent of the calories in the form of fat, and though the diet was undoubtedly deficient in vitamins A, C and D, practically no dental caries was seen.[37] The significant fact was noted that the boys could "only buy as a relish not sweets but fresh fruit.[38] Contrariwise, in "A Study of the Dental Problem in Hawaii," where mothers, children and food can hardly fail to have ample solar irradiation, we find that "cod-liver oil, whether vitamin tested or not, is administered by the gallon in some schools, yet the younger children suffer from defective, poorly calcified teeth."[39] This is confirmed by an investigation on "Dental Disease in Hawaii," by Drs. Jones, Larsen and Pritchard,[40] who find that "sun-

35. Bunting, R.W.; Crowley, Mary; Hard, Dorothy G., and Keller, Mary: Further Studies in the Relation of Bacillus Acidophilus to Dental Caries, Dent. Cosmos, 70:1009 (Oct.) 1928.

36. Press & Circ., Jan. 8, 1930, p. 17.

37. Mann, Corry: Proc. Roy. Soc. Med., Sec. Epidemology, Nov. 28, 1930, p. 229.

38. Lancet, Dec. 6, 1930, p. 1247.

39. Millberry, G. S.: A Study of the Dental Problem in Hawaii, p. 122.

40. Jones, M. R.: Larsen, N. P., and Pritchard, G. P.: Dental Disease in Hawaii, Dent. Cosmos, 72:699 (July) 1930.

shine, cod-liver oil, eggs, orange juice, tomato and milk—both human and fresh unpasteurized cow's milk—in good measure, singly or combined, do not prevent the rampant disintegration of the teeth of infants and small children. . . ." Here, too, in England, "we are already meeting with cases where children are devastated with caries, who have been dieted with cod-liver oil, and where the fond and anxious parents have pinned their faith to this food—in vain."[41]

Again, the "eat more fruit" campaign has been wonderfully successful and the substitution of fruit for sweets in families as well as institutions has revealed to many how much these natural foods excel the manufactured, at least with regard to the hygiene of the mouth and teeth. I do not think the "drink more milk" slogan is likely to have any continued success. Unlike some milk products, milk is expensive and dangerous. Further, experimental evidence has been brought forward which shows that milk is not by any means protective against caries, for to quote from a recent book with regard to Louise O. Kappe's study, it was said that "surprisingly enough, the children with the poor teeth actually drank more milk than those with the good teeth."[42] (Facts are surprising only when they prove that preconceived ideas are erroneous.) It was admitted that in the "poor group the diets containing much milk also contained much carbohydrate (cereals, potato, sweets and candy) and little vegetable and fruit."[42] They thus demonstrated what we always knew, that milk with all its vitamins is perfectly useless in preventing dental caries. Again, knowing all the time that vision was obscured by the vitamin fog, individual writers have not overlooked the importance of the detergent diet theory of the prevention of caries. A number of books, particularly in this country, on the feeding of children, have, during the last twenty years, been published, each in its own way emphasizing the benefits of a recognition of the detergent diet theory, while certain encyclopedias[43] contained numerous articles on various subjects associated with the teeth which kept as strictly to the dietetic doctrines I advocate as anyone could wish for. Similarly, individuals holding positions of authority, such as the editor of "The Modern Health" books, Prof. D. Fraser Harris, physiologist, made sure that the subject would be elucidated on what has been for many years recognized as the official and orthodox teaching of the dental profession in this country at least. Indeed, well considered and authoritative information is now being broadcast, not only throughout Britain but also throughout the whole civilized world.

Let me refer to two of the most important of these. You will remember an address by Sir Francis D. Acland which was broadcast a year or two ago and issued at the request of the Dental Board of the United Kingdom. This, in addition to being published in the ordinary way, was produced in the form of a gramophone record by the International Educational Society. The whole address is simple, straightforward, wholesome instruction, well suited for its purpose. Here, I can only refer to what is said with regard to diet and eating. He says, "Teeth must have exercise, particularly while the jaw is growing. A little child

41. Dent. Surg., Vol. 27, April 5, 1930.

42. Blunt, Katherine, and Cowan, Ruth: Ultraviolet Light and Vitamin D in Nutrition, University of Chicago Press, 1930, p. 66.

43. Harmworth's Universal Encyclopedia; Household Encyclopedia, and Home Doctor.

with only four teeth should be given hard crusts of wholemeal bread to chew now and then. From childhood onward, no one should ever end up a meal with anything that leaves a sticky paste on the teeth like cake, buns, pastry or sweets. As to sweets, a doctor went the other day to one of the islands of the Hebrides, and gave lectures on the care of the teeth in the schools, and he found that the only place where the children's teeth were bad was a village where there was a sweet shop! Finish a meal with oatcake or wholemeal bread with plenty of butter, or better still with fruit or uncooked vegetables, such as lettuce—and an apple is best of all. Never, never should a child have a biscuit or chocolate or anything else when it has gone to bed for the night."[44] I sent the full address as it was published to a friend who has been taking an interest in the subject for many years. His comment on it was, "Where have the vitamins fled?"

44. Acland, F. D.: Your Teeth and Your Health, Scot. Health Mag., 2:60-61 (June) 1930.

The other authoritative epitome of our views to which I would refer is the leaflet brought out by the Society of Medical Officers of Health, and now also issued by the People's League of Health. This was prepared by a committee, elected by the council from the various groups of the society. Though the committee was appointed when the vitamin craze was in full blast, a little quiet occasional discussion, continuing for about two years, interrupted by an incident resulting from the existence of the Medical Research Council that seemed likely to wreck the whole affair, brought the committee to unanimity. If other individuals would similarly sit down to a little quiet consideration of the facts of which we are already possessed, there would, I think, soon be an end to the school of thought which has during the last dozen years militated against the diffusion of knowledge so urgently required for the prevention of the diseases of the teeth and all the troubles of concomitance and consequent origin.

THE FUTURE OF DENTAL PRACTICE*

By A. M. SIMONS, B.L., Chicago, Ill.

ONLY fifty years ago, practically all care of the health was reserved to private practitioners. In 1883, Germany transferred a large section of the medical care of the German working class to social insurance organizations. This insurance movement has since swept on with accelerating speed from nation to nation until today medical practice in working class circles in a majority of countries outside the United States is wholly dominated by social insurance systems.

This movement not only grows geographically, but also within each country more and more of the population are brought within its control, and its scope expands to embrace each new professional

*Read at the Midwinter Clinic of the Chicago Dental Society, Feb. 3, 1931.

division of medical care and to increase the extent of its benefits.

No sooner does any form of care of the human body establish itself scientifically than it is brought within the scope of this system. The very fact that it is so included is a testimony to its scientific advance. German dentistry, in 1883, was only beginning to break loose from the physicians. As soon as it had accomplished this division and demonstrated the value of oral hygiene, prosthetics and dental service, it was caught up in the sweep of the insurance system.

Even in 1911, when the social insurance movement reached Great Britain, dentistry was made only an "additional benefit," but soon the clamor among the insured for dental service forced constant expansion until, but a few months ago, it was made a very vital part of the scheme. While it is not strictly "statutory" in the sense that every insured person is entitled to dental benefits, a practical effect of the latest regulations is to accomplish something very close to this object.

The dentists have been fortunate in that, until the present time, they have been able to stand back and profit from the physicians' experience in testing out all forms of insurance systems. The dentists could usually depend on the rule that what came to the physicians would come to the dentists within the next few years.

Because the physicians were slow to learn that the time to fix the form of an insurance system to meet professional standards is during the creative stage, the whole history of insurance legislation is filled with the lamentations of medical associations over their neglect to act in time.

Once the law is on the statute books, it sets up the most extensive social institutions created by any form of legislation. Now, social institutions are stubborn things. They are rooted deep in vested interests, customs, prejudices and legal rights. Once they have struck these roots into the soil, it is hard indeed to move them. On the other hand, when such legislation is first proposed, no territory is the undisputed property of any interest and the common good can be considered in setting up the institutions. We might well echo the statement of the official spokesman appointed by the British Columbia Medical Association to express the opinion of that organization before the Royal Commission investigating the subject of health insurance, when he says[1]:

> We do not want the history of health insurance in England to repeat itself here. There the medical profession was taken by surprise and suddenly saddled with an ill-considered political scheme, and while it is to their credit that they have loyally done their best to make it a success so far as this could be done, yet they felt that they were unfairly and discourteously treated. Probably it was to some extent their own fault. We must see to it that such a fault will not be ours.

In Germany and Austria and in the countries which, like Poland and other central European nations, adopted their systems from that of Germany, the medical profession finds itself so completely deprived of power by the original legislation that it has never been able to obtain the position of professional authority and dignity in relation to insurance practice to which it is entitled and which would secure the best interests of the suffering public. The dentists of these countries have shared the lot of the older branch of the medical profession. They have seen the fees for insurance practice

1. MacDermott, J. H.: Canadian Doctors and Health Insurance, Labour Gazette, May, 1930.

reduced to the subsistence point and then much of even this poorly paid practice drawn away to insurance society clinics conducted by factory methods.

Notwithstanding the frequently expressed regrets of British medical authorities at the lack of preparation and vigor at the time of the law's enactment, there was sufficient professional influence to establish a series of defenses which have not only prevented further encroachments on professional rights and duties by lay organizations, but also have formed a base from which dentists as well as physicians have lately been able to conduct a campaign that has resulted in further important gains.

Before describing these conflicts, it might be well to outline the issues and the position of the contestants. Insurance in all countries started, as it long ago started in this country, through fraternal, commercial, mutual, plant or other associations. Benefits at first were in cash. Then, it was found that medical treatment by restoring the invalid to health brought a great economy in total expenditures. The resulting extensive system of health insurance with medical care was confided to the management of the old societies, which confused cash and service treatments.

This subjected the medical professions to the lay control of societies and started a struggle that is just now climaxing in a threatened collapse of the entire original system of German social insurance. Not that it is really probable that this threat will be carried out. Drastic reforms have been introduced and greater ones will follow before Germany will go back to individual care of the health of her citizens.

When the dentists were taken into these systems, they had to accept the conditions which had already been created for the older branches of the healing professions. Recently formed systems have shown the very rare characteristic in social legislation of being able to profit by the experience of other times and places.

Nevertheless, the extension of the British system in the field of dentistry led the editor of the *Mouth Mirror* to an expression of the same sort of useless regret that had come from the physicians of almost every country in which a form of insurance had been adopted:

> The Dental Board as at present constituted was thrust upon the profession simply because, at the passing of the 1921 Act, the profession didn't bother about anything so long as an act of some sort was being passed. Apathy, laziness, laisser faire, or whatever one likes to term it, and a large dose of internal party difference, restrained the profession from asserting itself and demanding from the legislature measures beneficial and secure—and independence.

The United States is today almost the only island left unsubmerged by the tide of insurance legislation—a tide that nowhere shows any signs of immediate ebbing. That such a tide may be directed, if not swept back, is proved by recent experience in Great Britain and France. In these countries, the intelligently directed efforts of the medical professions have successfully defended the interests of patients and practitioners.

The flood of social legislation is at this moment rising toward probably unprecedented heights in the United States. This succeeds a temporary recession which followed the high tide of 1918-1920, when eleven official state commissions investigated, and six endorsed, compulsory health insurance. The movement for social legislation always ebbs and flows with the economic cycle, each period of depression being followed by a flood of such laws.

If we recall the "Greenback" and "Granger" legislation after the panic of

'73, the railroad regulation culminating in the Interstate Commerce Law and Commission after the "hard times" of 1884-1886, how populism, "Bryanism" and "muck-raking" came immediately after the deep depression of 1893-1895, how the "progressive" movement succeeded the cyclic drop of 1907 and, finally, how the economic "low" of 1914 had the Great War, with its mass of social legislation, including the health insurance movement just mentioned, as its aftermath, no crystal globe nor prophet's mantle is required to foretell what the immediate future will bring forth.

In fact, the full sweep of the agitation that precedes such social legislation is already upon us. Jan. 25, 1931, the governors of seven northeastern states met at Albany, on the invitation of Governor Roosevelt of New York, to discuss unemployment. The conference unanimously agreed to urge on the legislatures of their various states the immediate investigation of unemployment insurance.

Let it be remarked parenthetically, but none the less emphatically, that there can be no unemployment insurance without sickness insurance. No system of relief can discriminate against physical, in favor of economic, causes of unemployment. Moreover this is just the road by which sickness insurance has entered in other countries, and in the same company. Unfortunately this is also the combination that brings evils and confusion into the whole system of sickness insurance.

The American Association for Labor Legislation, the same organization that led the fight for social insurance in 1918-1920, has had a committee working for nine months on the draft of a model law. During the next few months, this bill will be introduced into a number of legislatures in addition to those represented at the Albany conference. In most of these, strong forces are now rallying to push such legislation to victory.

Workmen's compensation legislation has already given this country a long start toward sickness insurance. Largely because of the indifference of the medical professions, this legislation has already set a somewhat undesirable pattern for the inevitable future development. Although about forty-four states have such laws, nearly half of which include "occupational diseases" within their scope, which involves provisions for extensive medical service, including dental care in a rapidly increasing degree, the medical professions have had little to say about creating or administering such schemes. So slight has been the share of these professions in this large field of health activity that it is lamented by the National Industrial Conference Board, a research organization maintained by large employers, which says:

> From the beginning, medical questions have been among the most important of all questions involved in the administration of these laws. In many cases, however, they have been given but scant consideration. Even today, after the experience of a decade in the operation of these laws, only 15 of the 42 states with workmen's compensation laws have physicians attached to the administering board in any official capacity other than that of examining or impartial physicians who render opinions on the physical condition of the claimant. Such physicians have no authority to consider other questions relating to the case. In half of the states having medical departments, the medical advisor devotes only a portion of his time to this work. In only one state is a physician a member of the administrative board.

There is ample proof that this indifference and inactivity of the profession has brought many disadvantages to the physicians and dentists who come within the scope of compensation systems. Unless this legislative pattern is broken, the extension of its scope through sickness in-

surance will greatly multiply these evils.

The results achieved by the physicians and dentists during the enactment of the French sickness insurance legislation last year, and of the British dentists during the revision of the dental provisions at the same time, prove conclusively that when these professions are organized for action, and have their activities directed by a knowledge of the social, economic and political principles involved, they can so shape insurance schemes as to make their operation far less burdensome to the professions and far more helpful to the public.

During the next few months the legislative mold will be formed into which will be cast much of the future of American dentistry. Whether, as in so many European countries, that mold will be shaped to fit the interests and the ignorance of politicians, welfare workers, insurance organizations and other lay forces, and therefore to cramp, confine and distort the lives and all the professional relations of the dentists of America, depends almost entirely on the way in which the professional associations meet the situation.

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Editorials

THE ONE THING NEEDFUL

At this season of the year, just as the annual influx of graduates from the dental schools has been completed and the state board examinations have been held and the candidates slated for licensure, there comes the natural reflection on the possibilities to the profession of this infusion of new blood. If the best traditions of dentistry are to be maintained, each class of graduates should bring with it an addition not only to the science and art of dentistry but as well to the cultural and ethical equipment of the profession.

Students are better trained today than ever before in preparation for their dental college course, their matriculation requirements having been raised from time to time so that at least the fundamentals of a liberal education have been gained before admission to the professional school. Not only this, but the curriculum of the dental course has been amplified in every direction till the training embraces a much broader and also a more detailed study of the various subjects than was the case even a single decade ago. Today,

a student's graduation implies an altogether better preparation for subsequent practice, as also it should connote a broader outlook on life itself than has heretofore been possible.

All of this is matter for congratulation, but there is one thing in addition which should be cultivated more assiduously than it is in the consciousness of every young man or woman beginning the practice of dentistry. This is the thing that we in a vague way term professional spirit. To define professional spirit is not altogether easy. It is embodied more in the essence of self-sacrifice and altruistic service than in the accumulation of money; more in the glory of contributing to the welfare of humanity than in the material gains to be had from our daily endeavors; more in the sweet solace of relieving suffering than in the rewards that men call tangible; more in doing for others than in receiving the fruits for ourselves.

And the pathetic thing is that so few of our recent graduates ever have the faintest conception of what professional spirit is. How could they? Most young men and women enter dental school with the sole idea that it is an honorable calling and one in which they may hope to earn a livelihood and achieve a fair degree of material success. Their aim is laudable enough, but, at this stage of their development, they have very little of the ideal of altruism in their soul. This comes, if it comes at all, only from the actual experience of meeting and mastering human suffering.

Not till one has encountered the drawn face of agony and seen the tenseness of pain in the lines of the countenance, and then noted these lines relax and disappear as the result of the ministrations of the professional man, does one get a real glimpse of the consecration of service. Nothing can so surely bring to the young graduate a realization of what professional spirit actually is as the experience of successfully relieving suffering. A man may achieve all the material gain possible in his career, and yet fall short of the real goal of his cherished destiny if he fails to cultivate or to acquire the essence of altruistic service as it is met in everyday professional life.

Today, we hear much of dental economics; and, make no mistake, in these days of financial stress and worry, there is ample need

of a close study of our material needs, but if this emphasis on dental economics is continued too long or is carried to too great an extreme, we shall ultimately lose sight of the very thing that is best calculated to give us a legitimate basis for advocating economics or for making its study logical and effective. To put it metaphorically, we shall kill the goose that lays the golden egg.

Unless our professional life is leavened with the sweet savor of charity and loving kindness; unless we exhibit in our attitude toward society the altruism of true professionalism in our daily lives; unless we show a willingness to sacrifice something wholeheartedly for the public good, we shall not get very far toward the goal of our economic success, nor shall we attain to a status in the public mind that will justify even a consideration of the subject of economics.

The reputation of dentistry was built not on material advantage or aggrandizement but on professionalism pure and simple, and sometimes it would seem that in these latter days there was grave danger that we were jeopardizing both our purity and our simplicity. Heaven grant that in the evolution of the possible fruitful days to come, we shall not lose sight of the essence of true professionalism. It is our ethical outlook that has distinguished us from mere tradesmanship, and the moment we trifle with that, we are lost. We may be assured that for the maintenance of our cherished birthright, true ethics is the one thing needful.

"INASMUCH AS YE HAVE DONE IT UNTO ONE OF THE LEAST OF THESE, MY BRETHREN, YE HAVE DONE IT UNTO ME"

In the course of his president's address, May 6, 1931, Dr. Frank A. Delabarre, of Boston, Mass., recited an occurrence that is worth recording. He says:

> There was an outstanding incident of the year's work, which renews our faith in humanity and the essential character of the men in our profession.
>
> Dr. Richard H. Norton's attention was called to the fact that an unknown dentist had died at the City Hospital alone and without the consolation of family

and friends. The deceased was not a member of our Society and was unknown to any of us, but Dr. Norton personally gave him a decent burial at his own expense. After very considerable time and effort, the family was traced and he found them almost destitute and unaware of his death.

A group of about sixty members, on learning the circumstances, felt that no one man should bear the burden, and their response to an appeal for funds was quick and generous. A substantial balance was turned over to the widow, who was overcome by the totally unexpected action, and was profound in her thanks.

Dr. Norton deserves the gratitude of all of us for his spontaneous and unselfish effort to uphold the dignity and traditions of our profession.

This was not only a splendid gesture on the part of those sixty men, but when "Dick" Norton bethought himself to do that generous act, and furthermore, when he actually did it, he was administering a better service to dentistry and to humanity than he could have done by the performance of the most finished piece of technical work, or the greatest surgical achievement in dentistry. To open up the human heart to the needs of suffering humanity; to succor the helpless and despairing who by circumstance or chance have fallen by the wayside; to show respect to the dead regardless of whether or not they are known; to be at one with the sympathies and the sorrows of one's fellowman, reaching out the helping hand in loving kindness, in charity and in unselfish devotion—all of this is better than to be acclaimed the most noted scientist in the world. The human heart means more than the fertile brain, and to do a kindly act is better than to win a victory on the battlefields of fame.

It was given to Dr. Norton to perform quietly, unostentatiously and whole-heartedly an act that must be classed as sanctified service, and when the deeds of men are recorded on the balance sheet of time, it is comforting to reflect that dentistry had a man like him. It was not only that his personal sacrifice meant so much but also that the entire profession was glorified thereby.

A CERTAIN OBLIGATION TO OUR JOURNAL

The wide circulation of THE JOURNAL naturally attracts to it a great deal of material on the part of contributors from all over the world, and to these contributors we acknowledge our deep

indebtedness. But we have a certain obligation that is sometimes not recognized. This publication is THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION. It is published and maintained by the Association and naturally our first obligation centers in the organization itself. It is of course quite flattering to have the material from other associations presented to us for publication, as well as to have volunteer papers of a high order submitted to our official organ, but we must not forget that, after all, our first allegiance is to the American Dental Association, and therefore the proceedings of our annual meetings must logically have the right of way.

In the past, we have at various times felt impelled to yield to the pressure brought for the early publication of outstanding papers read by other organizations or volunteered without having been read, with the result that there has been a hardship on account of the delay in the publication of our own papers.

But let it be said in this connection that frequently delay has been occasioned by failure on the part of essayists to properly prepare their papers, and to complete them for publication. It is a common occurrence to receive a paper with some of the illustrations missing, or to be given instructions to hold a paper for better revision—a revision that sometimes never materializes. There are numerous delays of various kinds not connected in any way with editorial policy, such, for instance, as the loss of time in preparing discussions that have been reported by a stenographer. These discussions are a source of greater dissatisfaction and delay than any of the material that comes to the editorial department. It is seldom that the report of a discussion is accepted by the speaker as being correct; and this does not refer to any one stenographer but to all of them. The only equitable and economical way of handling discussions in our section meetings is to require that speakers prepare their discussions in advance and have them typewritten. The cost of reporting the discussion of the section meetings at Denver, according to our Business Manager, amounted to $1,236.50, and this is an entirely needless expense. A rule should be made that in case of an outstanding impromptu discussion, the

speaker be required to furnish a typewritten copy to the chairman or secretary of the section before he leaves the meeting. With a rule that discussions are to be prepared in advance and turned in at the session at which they are presented, and that an impromptu speaker is required to have his talk typewritten, there will be little delay in getting out our proceedings.

And the fact should again be emphasized that our own transactions have prior claim on our consideration, and in the event that there is space left after provision is made for this material—which is always the case when it is properly condensed—the really worth while outside material may then be welcomed to our pages.

EDWARD K. WEDELSTAEDT

(1857-1931)

Edward K. Wedelstaedt was born March 25, 1857, in St. Paul, Minn., and died there, June 2, 1931, having lived in St. Paul all his life.

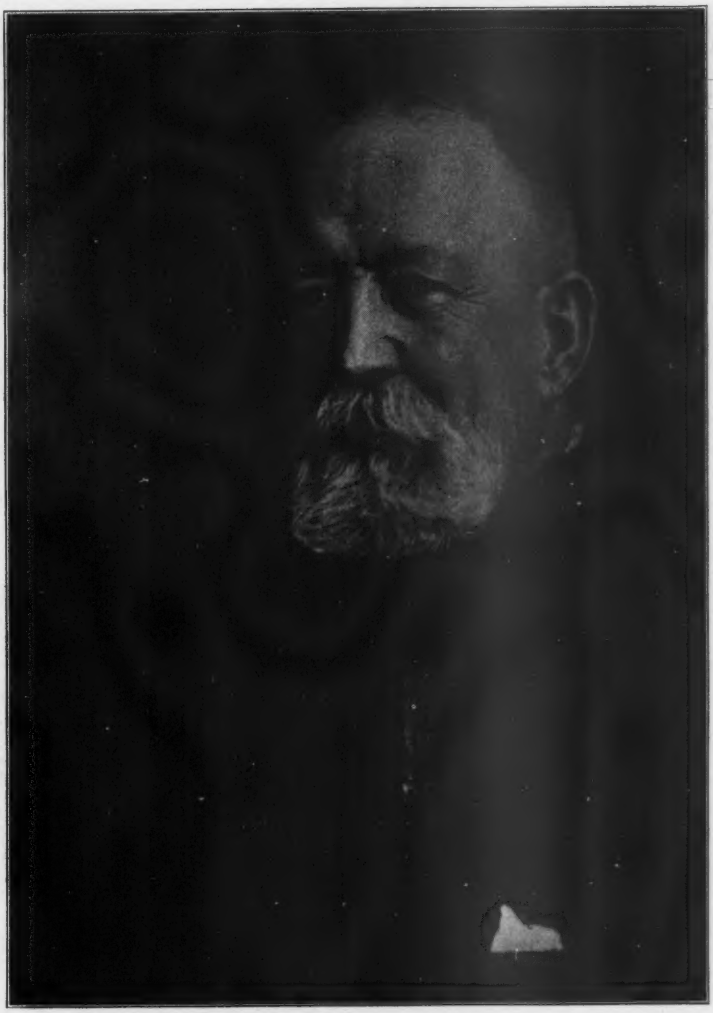
He began the study of dentistry under a preceptor, and, at various times, he received instruction from some of the most noted men in dentistry, chiefly and for many years under the late G. V. Black of Jacksonville and Chicago.

Exhibiting at an early stage of his career a unique ability in the science and art of dentistry, he soon became an outstanding figure in the development of the profession. Born to leadership, he organized in 1896 the G. V. Black Club of St. Paul. which is still in existence. This organization holds monthly meetings, and it is typical of the spirit infused in its leader to note that some of the men come from hundreds of miles each month to attend these meetings. For many years, Dr. Wedelstaedt and his loyal band of less than twenty members organized a midwinter clinic that drew distinguished men from every section of the country, extending from coast to coast. It was the first of the great outstanding midwinter clinics that have now become so popular.

Into this work, Dr. Wedelstaedt threw his heart and soul, and no man was more influential than he in promulgating the theories of Dr. Black. This organization work soon extended till study clubs were formed in the northwest as far as Seattle, and to Dr. Wedelstaedt must be accorded the credit for bringing to the attention of this whole region the most advanced ideas in scientific dentistry. His influence for good can never be computed, and dentistry is a better profession today because he lived and labored for its advancement.

Dr. Wedelstaedt is survived by his widow, Mrs. Alice Humbird Wedelstaedt, and two brothers, both physicians, Dr. Mark Wedelstaedt of Long Beach, Calif., and Dr. George Wedelstaedt of Los Angeles.

The remains were cremated in accordance with his wishes.



EDWARD K. WEDELSTAEDT
(1857-1931)

J. LOWE YOUNG, D.D.S.
(1868-1931)

J. Lowe Young was born in Eckford, Middlesex Co., Ontario, Canada, Feb. 10, 1868; died May 23, of heart disease.

Dr. Young attended the Toronto Dental College, and the Philadelphia Dental College, where he was graduated in 1890. After practicing his profession in Montana for three years, he went to Detroit, where he continued the practice of dentistry until he took the Angle course in St. Louis in 1903 and became one of the teaching staff. He had previously been a member of the faculty of the Detroit Dental College. In 1905, he came to New York, devoting his time thereafter exclusively to orthodontia. When the school of dental and oral surgery was organized, he was appointed professor of orthodontia, but his health forbade his continuing in that post.

He served as president of the American Society of Orthodontists, the New York Society of Orthodontists and the Eastern Association of Graduates of the Angle School of Orthodontia as well as of the First District Dental Society of New York. He was a Fellow of the American College of Dentists and the New York Academy of Dentistry and a member of Delta Sigma Delta Fraternity.

Dr. Young was a man of positive convictions, yet one who welcomed information and valued every contribution to the science of orthodontia. His frankness and sincerity made him highly esteemed.

He contributed generously to his specialty by literary contributions, clinical demonstrations and many useful inventions. His advice was often sought and generously given and in a fashion which characterized him as a born teacher.

Dr. Young is survived by his widow, Mrs. Hester Bateman Young, three daughters, two sons and a grandson.

GEORGE EDWARD HARDY, D.D.S.
(1868-1931)

George Edward Hardy was born in Lunenberg County, Virginia, July 12, 1868; died, May 19, at his home, 518 Cathedral St., Baltimore, Md.

Dr. Hardy graduated from the Baltimore College of Dental Surgery in 1888. He continued his studies at the College of Physicians and Surgeons, graduating in medicine in 1889. He became associated with Dr. Richard B. Winder, commandant of the Andersonville (Va.) Federal Prison during the Civil War, whom he succeeded in practice.

Dr. Hardy was clinical instructor in the dental college from shortly after his graduation until he was appointed professor of physiology, a chair which he occupied until about 1923. He was a member of the dental staff of the Johns Hopkins Hospital for many years and a member of the board of the Henry Watson Children's Aid Society.

Dr. Hardy was a past president of the Maryland State Dental Association and he had twice been president of the Baltimore City Dental Society. The widow, three daughters, a son and two grandchildren survive.

J. LOWE YOUNG, D.D.S.
(1868-1931)

BUREAU OF DENTAL HEALTH EDUCATION

A REPORT ON DENTAL CARIES IN TEN THOUSAND FOUR HUNDRED FORTY-FIVE UNIVERSITY STUDENTS (1928-1930)*

By P. J. BREKHUS, B.A., D.D.S., Minneapolis, Minn.

CONFIDENT that the last word on dental caries has yet not been said, although volumes have been written and a great mass of reports on serious investigations have been published during the past century, I am presenting some clinical findings relative to this mysterious disease.

There is nothing new in the picture I am about to present—it is only an accumulation of facts observed in every-day practice, but in the act of accumulating these facts, certain independent reflections have been stimulated, and in presenting these independent reflections, it is my hope to provoke some interchange of ideas.

As a supplement to an investigation of mouth conditions in 10,000 patients from the clinic in the College of Dentistry of the University of Minnesota, a report of which was published by the University Press in 1928 under the title, "The Loss of Human Teeth," a survey of the dental condition of the incoming freshman students has been conducted in conjunction with the complete physical examination which every freshman student has to take before entering the university.

The former was a mixed group, social, economical and educational, the majority coming from the so-called lower class, as institutional patients often do. The ages of these patients ranged from 10 to 75 years. The question naturally arose as to whether these findings presented a true picture of the average.

The university students (average age, 18) as a group represent those of our citizens who are in a position to take advantage of the best that modern life has to offer in the way of prevention and cure. These young people have grown up during a period when educational propaganda on diet, oral hygiene, the importance of frequent dental examination and better dental care has been freely disseminated. The fear of focal infection has also been a factor in arousing greater concern in parents about their own and their children's teeth. The statistics here presented should therefore give a fair index of the degree in which dentistry has succeeded in preventing dental caries.

On the charts (Figs. 1-2) are recorded the dental conditions of 3,711 persons examined in 1930. The upper half of the chart represents the upper jaw

*Read before the International Association of Dental Research, Memphis, Tenn., March 22, 1931.

and the lower half represents the lower jaw, the center representing the median line. The figures 1-7 to the right and left under each column represent the teeth from the central to the second molar on both sides above and below. The third molars are not included in this tabulation for the reason that, in a great many instances, the third molars are not erupted in a group of persons of this age; and it is impossible without the aid of roentgenograms to ascertain whether the teeth in question are extracted, unerupted or congenitally missing. In these columns are tabulated on percentage basis: (1) teeth extracted; (2) teeth with active caries and no fillings; (3) teeth with fillings: (*a*) amalgam, (*b*) gold, (*c*) silicate and (*d*) cement; and (4) teeth not affected by caries.

TABLE 1.—PERCENTAGE OF PERFECT MOUTHS

	1928	1929	1930
Male	4.70	2.55	1.58
Female	1.80	.81	1.28

TABLE 2.—PERCENTAGE OF FILLING MATERIALS USED

	1928	1929	1930
Amalgam	73.0	72.76	72.17
Gold	17.3	17.62	17.36
Silicate	8.2	8.17	8.89
Cements	1.5	1.45	1.58

The findings were as follows: In 1928, 3,346 students examined showed 34,384 affected teeth; in 1929, 3,388 students examined showed 33,697 affected teeth; in 1930, 3,711 students examined showed 38,283 affected teeth; total number of students, 10,445; total number of affected teeth, 106,364. Of these affected teeth, 8,958 had been extracted, 13,674 showed active caries and the remaining 83,732 contained fillings. This gives an average per person of slightly more than ten teeth affected by caries, and per person less than one tooth that had been extracted.

The percentage of perfect mouth conditions was very small and seemed to be on a decline. (Table 1.)

It is interesting to note how the percentage of the several filling materials employed corresponds in the three different years. (Table 2.) This may be of minor importance in this study, but it does indicate certain fixed habits in the profession regarding treatment of caries.

To me, the most significant thing brought out in this study is the varying degree of susceptibility to caries in the different teeth. In comparing figures, we find it convenient to divide the twenty-eight teeth into seven groups, which brings out the results shown in Table 3. (Fig. 3.)

TABLE 3.—VARYING DEGREES OF SUSCEPTIBILITY IN DIFFERENT TEETH

Group	Positions of Teeth	Susceptibility Per Cent
1.	$\frac{6}{6}$	95
2.	$\frac{7}{7}$	75
3.	$\frac{5}{5}$	45
4.	$\frac{4}{4}$	35
5.	$\frac{2.1}{2.1}$	30
6.	$\frac{3}{4}$	10
7.	$\frac{3.2.1}{3.2.1}$	3

We find a varying degree of susceptibility in the different teeth ranging from 3 to 95 per cent. Many reasons for this variability, the principles of which are anatomic or developmental variations, have been brought forth, but as far as I can find in checking over the dental literature, they have been based on clinical observation, not on laboratory investigation. And, in my opinion, here is a field for much profitable research on dental caries. The necessary equipment and lab-

oratory facilities to carry on such an investigation have been provided for by Dean Lasby from funds made available by the administration of the general research budget at the University of Minnesota, and we hope, at some future meeting of this organization, to be able to report whatever progress shall have been made.

It would be interesting to enter into an analytic discussion of the different factors generally considered to be involved in the problem of dental caries, such as diet, mouth hygiene, action of saliva, form and structure, time of eruption, periods of susceptibility and immunity and sex differences. The time allotted for this report does not permit this; but I do want to state my conviction that the sum of definite knowledge concerning this disease, added to what was already known and observed by scientific men almost a century ago, is very small.

To substantiate this conviction, I shall quote at some length from Nasmyth's researches on the development, structure and diseases of the teeth, published after his death in 1849. From page 215 of his book,[1] I quote:

> In regard to the development of the process of decay, it appears to me that in most cases it is a combination of vital and chemical processes. It cannot be considered exclusively under the influence of either, but at the same time I consider it more the effect of vital than chemical action. Without any deficiency in the original organization of the tooth, I consider it quite possible for a faulty vitality to originate decay; but the simple presence of food, without any natural deficiency in the tooth, I consider incapable of producing that effect.

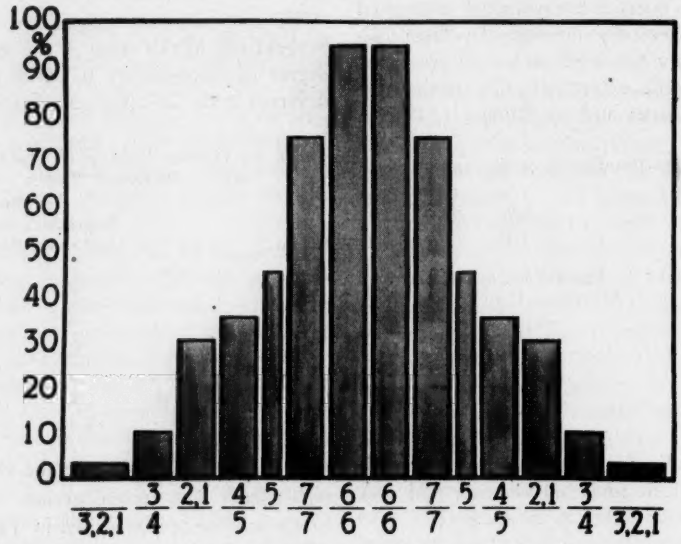


Fig. 1.—Comparison of susceptibility to caries in the different teeth (from a study of dental caries in 10,445 university students.

1. Nasymth, Alexander: Researches on Development, Structure and Diseases of Teeth, London, 1839.

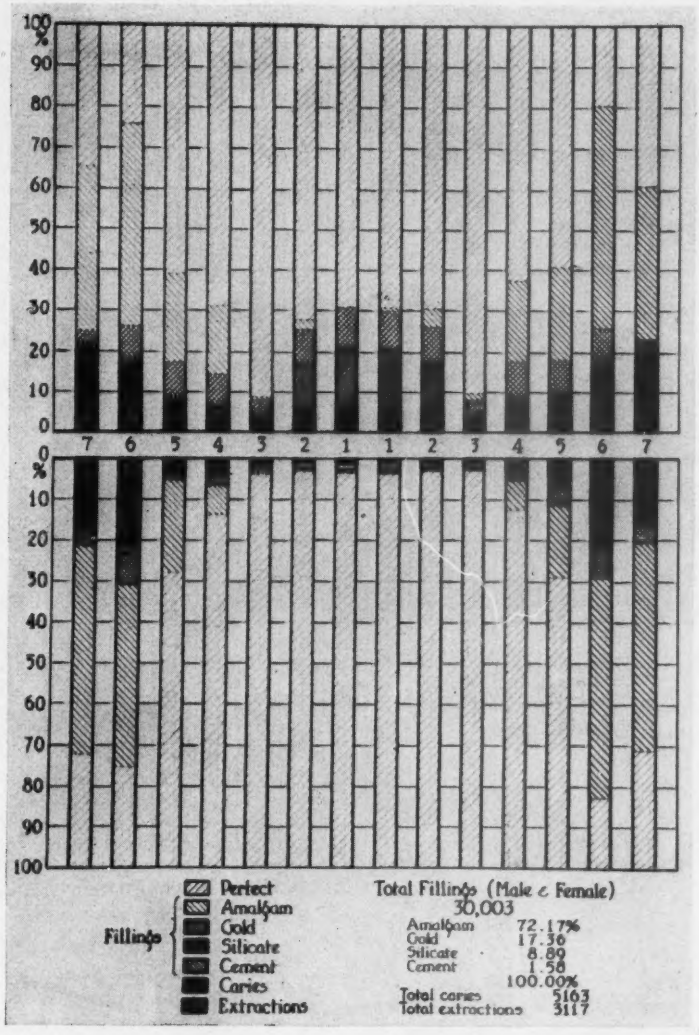


Fig. 2.—Dental condition of 2,151 male freshman students (University of Minnesota, 1930).

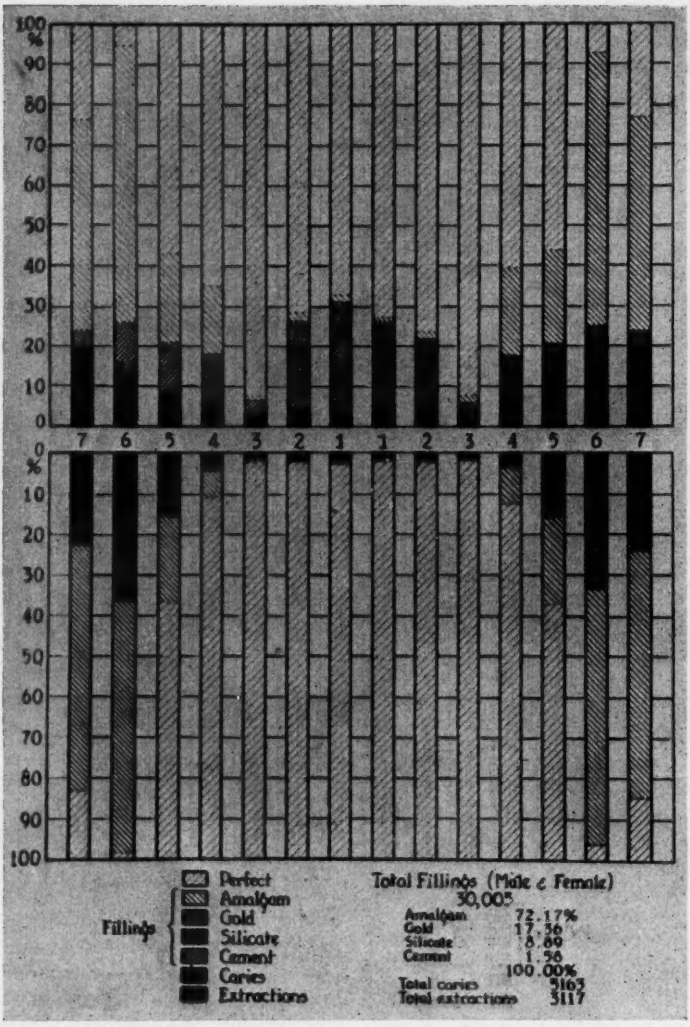


Fig. 3.—Dental condition of 1,560 female freshman students (University of Minnesota, 1930).

In the remainder of this paragraph, he goes on to explain his theory of this "vital and chemical action." He then continues to compare his theory of dental nutrition with the problem of general nutrition:

> This subject, in my opinion, requires a deeper consideration than at first sight we might be led to give it, on searching for the sources of decay. In the first place, the vital processes of the animal economy are continually carrying on a universal change in the whole system. The materials of the body are in a constant condition of progression or replacement by one process or another, and a supply of new material is constantly going on. When these processes are balanced, the functions of the part are perfect; but if there be a subtraction of material without an adequate supply, decay of the part must succeed. We have in the tissue of ivory a cellular structure, furnished with organized animal matter, in the presence of the rows of persistent nuclei, surrounded by the parcels of earthy matter which form the characteristic of the structure. We have, therefore, analogous constituents to other tissues, where the processes of replacement or absorption of old, and nourishment of new parts goes on; we cannot, then, deny this tissue the exercise of similar functions. If the absorption by the system goes on to a greater extent than the renovation, we must have a consequent deterioration of healthy constitution in the structure. Decay will be developed in the different portions of the tooth in an inverse ratio to the power of resistance. The more perfectly the structure is organized, the greater will be the power of resistance. I have pointed out the deficiencies in structure which occur, so far as I have been enabled to notice them.

With exception of the bacterial theory advanced by Koch and Miller in 1884, what new has been added to our stock of knowledge regarding this disease? With the absorbing interest in mechanical repair and replacements, the dental profession had almost forgotten that it was dealing with symptoms of a "vital process"; and when Dr. Bödecker[2] and others have called attention to this theory, it has been regarded as a new thought, although Nasmyth had explained it beautifully ninety-five years ago.

As to the congenital faults supplying a mechanical source of decay, Nasmyth goes on to explain:

> The interrupted appearance of the nuclei composing the fibre; the masses of animal matter distributed throughout the substance of the ivory; the deficiency of cohesion noticed betwixt the enamel and ivory; the peculiar cellular appearance resembling vesicles, with no appearance of nucleus or fibre. In fact, any deviation from the normal structure of perfect ivory seems to predispose to natural decay. These congenital faults supply a mechanical source of decay, by allowing the permeation of the fluids of the mouth, mingled with food, to enter through any minute orifices generally present in the undulations of the enamel. The fluids and food become stagnant in these cavities, get putrid and encourage a like process in the animal matter of the ivory around, and thus deprive that portion of the tissue of its vitality and support.

Dr. Hyatt,[3] with his predecessors and followers, advocates that these congenital faults be overcome by prophylactic odontonomy. I think the profession should agree to this.

The importance of oral hygiene and correct diet is brought out very lucidly by Nasmyth when he states:

> In cases of lower vital resistance introduced by presence of a foreign body, such as may be used for false teeth, then I know it is possible (teeth will decay), and unless great care and cleanliness be practiced, that effect is sure to follow.

How could the importance of correct diet be more clearly stated than in the following: "We have therefore, analogous constituents to other tissues, where the processes of replacement or absorption of old and nourishment of new parts

2. Bulletin of Information, Columbia University, Oct. 25, 1930, p. 11.

3. Hyatt, T. P.: Review of Dental Literature of Pits and Fissures, J. Dent. Res., 10 (Dec.) 1930.

go on; we cannot, then, deny *this* tissue (the teeth) the exercise of a similar function."

Throughout all times, teeth have decayed, have been filled, have been extracted, have been replaced by partial and full dentures in greater and greater numbers as civilization has advanced and the professions have become more scientific. Evidently there is yet room for research.

A NEW ASPECT OF PROPHYLACTIC ODONTOTOMY*

By THADDEUS P. HYATT, D.D.S., F.A.C.D., and H. SHIRLEY DWYER, D.D.S., New York City

EMERGING from the gloom of obscurity and insignificance, without any of the mystic trappings and ancient traditions surrounding our brothers of the medical profession, dentistry, as a profession, has risen rapidly to its present position. Is it possible that this humble origin still casts a shadow over our writings? For over a hundred years, we have been blushing ruefully and apologetically before health workers, our medical friends and the public, not only admitting that we do not know the cause of the very disease we, as a profession, claim superior ability to diagnose and to treat, but also publishing article after article emphasizing this lack of knowledge. True, there are too many conditions involved to fix the cause of caries on any one definite factor, but many papers refer to the cause of caries as absolutely unknown.

Can we not understand the impression and the effect that this attitude has had on the medical and the lay mind? Have we so little imagination that we utterly fail to understand why, for so many years, dental lesions have been ignored as one of the important factors in public health? Does this not help to explain the slowness of the adoption of any scientific procedure, or of any practical program for the prevention of caries as a public health measure?

How can we prevent that which we do not know the cause of?

Because of this attitude, this mental apathy, this lack of recognition of facts and truths, the scientific health value of dental service has not yet received full recognition as an important and necessary part of all public health work. This is shown by the almost insignificant appropriations made for this work by state and municipal officials.

Of late, many of us have been sitting spell-bound at the feet of laymen who preach dental economics. Why? Because of that inferiority complex which says, "I don't know." Why not consider what we do know?

Permit us to state some facts or axioms which we believe none will dispute and which every member of our profession knows or should know.

An axiom is defined as "a self-evident or necessary truth; hence, any proposition that men universally accept," and it has been said that "axioms are the foun-

*Read before the Section on Mouth Hygiene at the Midwinter Clinic of the Chicago Dental Society, Feb. 4, 1931.

dations of science." We therefore intend building our suggested preventive procedure on certain well known statements:

1. The acidity of the saliva is never sufficiently strong to cause decay. (G. V. Black.)
2. Decay does not start from within the tooth and work outward (G. V. Black.)
3. The undisturbed retention of food débris creates a condition favorable to caries development. (Robert Arthur; Charles F. Bödecker.)
4. For caries to progress in dentin, there must be an opening to the surface. (G. V. Black.)
5. There is a distinct difference between the progress of decay on smooth surfaces and decay in enamel defects. (C. F. Bödecker.)
6. The structure of the tooth governs the rate of the progress of decay, but does not affect the liability to decay. (R. W. Bunting.)

These principles constitute the foundation on which is based the operative procedure we advocate for the prevention of a large percentage of carious cavities. Please understand that this preventive procedure is not offered as a panacea for all dental woes. It is a simple, practical and scientific operation which will greatly reduce the number of more complicated and dangerous conditions, and is used as a preventive measure.

Are these axioms? Are they correct and scientific? If we know these simple facts are true, why not proclaim to the world at large the things that we *know?* Let us stop publishing, broadcasting and telling our audiences what we do *not* know. It belittles the intellectual standing of any group of men to be constantly saying, "I don't know this," or "I don't know that." Let us tell the world what we do know. We do know that these statements are truths; we do know they are supported by facts. Then let us broadcast this knowledge, that the world may begin to have more confidence in and a greater appreciation of our intelligence, because we do know something.

Our suggestions for a preventive operative procedure which we believe will reduce the percentage of carious cavities by from 50 to 70 per cent are based on (1) the knowledge which our profession already possesses as stated in the six axioms quoted; (2) the statistical data of actual conditions secured from a careful examination of 12,753 persons of all walks of life between the ages of 17 and 60, and (3) our scientific knowledge of the growth and development of the tooth before eruption.

Before determining an operative preventive procedure, let us consider what it is that makes such operative measure necessary. Let us begin at the beginning—the development of the tooth. We know that the crown form of the tooth develops from certain definite primary lobes which unite in a variety of combinations to form the different teeth. With a few exceptions, the crowns of the teeth are found to develop from four primary lobes variously arranged in the different anatomic forms. In considering the developmental anatomy of the teeth, we must think of each of these lobes as a separate entity eventually fusing with the other lobes into a complete whole. From this point of view, we will readily see how each lobe may present a number of surfaces. The fusion of these separate lobes, being along their contiguous surfaces, which we choose to call their coalescing surfaces, is recognized externally by a developmental line. This differentiation in terminology between coalescing surfaces and developmental lines is used for the following reasons: 1. A line has length

without breadth. 2. A surface is a plane which has both length and breadth. Therefore, the coalescing surfaces, having both length and breadth, cannot be classed as lines.

When these lobes have finally merged, the coalescing surfaces are no longer discernible and leave but a line to indicate their presence. Thus, we have the developmental lines as the external indication of the fusion of the primary lobes. These external evidences of union, or what are called developmental lines, vary in number in the different teeth in direct proportion to the number and arrangement of the primary lobes.

In enumerating the developmental lines as indicated on the various teeth for statistical purposes, we have endeavored to constrain ourselves to the minimum number compatible with common sense. To quote parenthetically at this point, apropos a statement relative to statistical study: "Statistics are no substitute for judgment." We shall, therefore, consider the developmental lines evidenced on the occlusal surface of the molar teeth as one ramifying developmental line rather than the five or six individual developmental lines which actually occur. Thus, we find that, in the entire adult dentition, we usually have 164 developmental lines indicating the union of the coalescing surfaces.

Now let us turn once more to the origin of these developmental lines. We have previously stated that, by the fusion of the primary lobes, the crown form is determined, and that the developmental lines are external evidence as to where this union took place. Undoubtedly, many factors enter into the proper coalescing of these lobes, and it is not our purpose to enter into this phase of the subject, but we do know that if all the essentials for proper coalescing are fulfilled, the resulting union should be so nearly perfect that the developmental lines would show no appreciable demarcation. Often, we find that some one of these variable factors has failed to perform its normal function, and along the developmental line there is an imperfect coalescence, leaving what is technically called a pit or fissure.

We have stated that there are usually 164 developmental lines in the adult dentition. Had we included all of the developmental lines found on the occlusal surfaces of the molars, the total would be increased to 202, but desiring to simplify our paper as much as possible, we have counted only 164 developmental lines. In the upper teeth, we find five such lines per tooth; in the lower second bicuspid and first molar, six, and in the other lower teeth, five.

There are only twenty-four tooth surfaces in the entire mouth on which we find no developmental lines. We find these developmental lines on the exposed smooth surfaces (such as the labial surface of the incisal teeth), the sheltered smooth surfaces (as the mesial and distal surface of the bicuspids and molars) and on the grooved surfaces (the occlusal surface of the posterior teeth, the lingual surface of the upper molars and laterals and the buccal surface of the lower molars). Considering them from this aspect, we find that we have seventy-six developmental lines on the exposed surfaces, forty on the smooth sheltered surfaces and forty-eight on the grooved surfaces. With this knowledge of the number and location of the developmental lines, let us compare the data with the record of where carious cavities are most frequently found.

We find that, in an examination of 12,753 persons of the ages from 16 to 60, 52.44 per cent of all the carious cavities

were found on the occlusal surfaces of the bicuspids and molars; 5.90 on the buccal surface of the lower molars; 4.44 on the lingual aspect of the upper molars, and on the lingual surface of the upper lateral incisors, the remainder of the cavities or fillings, 37.22 being distributed over the other surfaces of the other teeth.

Again let us refer to the development of the tooth. We mentioned the fact that not infrequently there was an imperfect union of the primary lobes, resulting in an imperfection in the coalescence surfaces externally recognizable in the majority of cases as a pit or fissure in the developmental line. These imperfections are largely confined to the occlusal surfaces of the molars and bicuspids, the lingual surfaces of the upper molars and lateral incisors and the buccal surfaces of the lower molars.

We now have three groups of observed data to compare. Our first inspection shows us the following:

1. The surfaces on which most caries occurs are the occlusal of the bicuspids and molars, the lingual of the upper molars and laterals and the buccal of the lower molars. 2. There are 164 developmental lines in the entire mouth, and of these, there are seventy-six on the exposed smooth surfaces; forty on the sheltered smooth surfaces, and forty-eight on the grooved surfaces. 3. The developmental lines are divided into two classes: those where perfect fusion most frequently occurs and those where imperfect fusion most frequently occurs.

All of which can be further condensed to the fact that the surfaces most prone to caries are the same surfaces on which we most frequently find imperfect developmental lines showing pits and fissures; and that these surfaces are the occlusal of the bicuspids and molars, the lingual of the upper laterals and molars and the buccal of the lower molars; and that there are almost twice as many carious areas on the developmental lines of these surfaces as are found on all the other surfaces added together.

What, then, is a common sense interpretation of these facts? For answer, we must turn back to the axioms.

We know (axiom 1) that caries is not caused by the free acid of the saliva; nor (axiom 2) has it started from within the tooth and eaten its way to the surface; but axiom 3 teaches us that in the restricted areas where food débris is retained, in the imperfections of the developmental lines known as pits and fissures, an acid of sufficient concentration is formed which breaks down the tooth structure. We believe this to be a sane conclusion, and in it we are supported by leading dental authorities, such as Professor Bödecker, who writes: "On no other surfaces of the teeth can food find a more lasting and effective retention than in enamel fissures." We might say in passing that over 400 years ago, a German writer made practically this same statement.

These are some of the things we know. We also know our operative ability and the means at our disposal to meet these conditions. Years more of research in biochemistry, on the endocrines and in diet, may bring forth new theories and new methods, but, in the interim, for the sake of the health of our nation's childhood, which governs the future progress of this country, we should unhesitatingly apply the knowledge that we already have.

The simplest, quickest, most comfortable operation in all the field of operative dentistry is the eradication of these enamel defects before they become carious. This may be accomplished either by immunizing or by filling when the imperfection penetrates to or almost to the

dentin. The operative immunization procedure is very simple, and, according to Prof. Bödecker, the method is "to eradicate the fissure by careful grinding and polishing so as to make it self-cleansing, thus preventing the retention of food." This eradication is best done by starting with a No. ½ round bur and using successively larger burs until the shape of the pit is changed to that of a shallow self-cleansing groove, when it should be polished. If the pit or fissure is found to penetrate through or nearly through the enamel, enlarging is stopped and the defect filled as any very small cavity would be. This is not a new process nor an untried theory. Dental authorities as far back as 1803 advocated this, and many of our leading teachers on down through the years have urged it and have practiced it. James Truman said: "I regard it as positive malpractice to allow any fissure to remain unfilled where the slightest evidence of softening of enamel exists." C. N. Johnson says: "An operator owes it to his patients not to overlook the slightest defect." Over a period of more than 100 years, our foremost teachers have been urging this care of the tiny defects—the pits and fissures—a simple little preventive operation which we know how to do.

May we call your attention to this fundamental fact: This entire paper is based on what we know—what our profession knows. The introduction, the data and the suggested operative preventive procedure are all based on what we know to be scientific facts, and what really constitutes the actual knowledge possessed by our profession today. We are not offering speculative or promising theories regardless of how logical they may seem. Time enough to change our practice when we have secured new knowledge, new substantiated data. We are offering to the members of our profession a simple operative preventive procedure which Professor Bödecker claims will reduce dental caries by over 50 per cent, and this too, we say, regardless of diet, regardless of what actually may be the cause of dental caries.

THE FIRST PERMANENT MOLAR*

By M. EVANGELINE JORDON, D.D.S., Los Angeles, California

PREPARING THE MOUTH FOR THE ERUPTION OF THE FIRST PERMANENT MOLAR

AS THE first permanent molars are the most valuable of the teeth, it is imperative to conserve them in every way.

As a children's dentist, I consider them the most valuable of the teeth, first because they are the first of the permanent set to erupt and they are in position for use six years before the second molars erupt; secondly, if they erupt out of position because of early loss of the deciduous molars, it is necessary to resort to orthodontia to restore occlusion, and thirdly, if they are lost early in life, no repair will ever compensate for this loss.

*Read before the Section on Mouth Hygiene at the Midwinter Clinic of the Chicago Dental Society, Jan. 14, 1930.

For this reason, conservative dentists should anticipate their eruption and strive in every way to assure each patient a perfect denture.

In preparing the mouth for the eruption of the first permanent molars, one of the most important duties of the dentist is to fill every deciduous tooth and keep it filled. Possibly there is no weaker link in the chain of dentistry than that one point. The average dentist has strange ideas about deciduous teeth, and I find many men placing temporary fillings in the second deciduous molars of 3-year-old children. A temporary filling in a tooth that is to serve for mastication nine years!

The average dentist does not know the anatomy of the tooth when he places a large restoration of silver in a deciduous molar and expects the pulp to survive. We must remember that, in proportion to the size of the tooth, the pulp of a deciduous molar is twice as large as that of a permanent one.

The mistakes of the average dentist range from telling a mother of a 2-year-old child that it is not necessary to fill the first teeth as they will soon be lost, to capping exposed pulps.

One of the most glaring mistakes is the filling of approximal cavities between the deciduous molars. One could not readily believe that a person familiar with the fact that a tooth in the jaw has movement would undertake to put in one filling to restore two cavities, one in the mesial surface of the second molar and one in the distal of the first molar. While fixed bridges may with safety be placed on permanent teeth, I doubt that such a restoration would ever be successful on deciduous ones because of increased movement due to the growth of the jaws. A filling between two teeth soon breaks away from one or both teeth and an abscess quickly results from the rapidly advancing caries, protected from mastication by the loosened filling.

Many such teeth have fallen under my care, and I recall one case in which all eight deciduous molars had pulp involvements under four highly polished silver fillings.

In my experience, which has covered twenty-five years, limited to children's work, there are very few exceptions to the rules (1) that copper amalgam is the only successful filling material for deciduous teeth; (2) that deciduous teeth with very deep cavities or exposed pulps already have infected pulps which must be extirpated to maintain the usefulness of the tooth, and (3) that at least 50 per cent of abscessed deciduous teeth may be successfully treated and continue in service until their normal time to be shed.

PROPHYLAXIS

Intelligent dentistry of today means prevention and the most valuable form of prevention is regular prophylactic care. I have the patient come first toward the end of the second year, examining and giving a prophylactic treatment, which is repeated every four to six months.

When the twenty teeth are in position, I have the patient return every three months until the appearance of the first of the permanent molars, when the prophylactic treatment is given every two months until the patient is discharged, at the age of 15.

You may say that a child under 2 years is too young to go to a dental office, but it is about that period that habits contributing to malocclusion may be formed; such as sucking fingers or other objects (even bed clothes), tongue thrusting, nail biting, bad sleeping postures and many other habits that will lead to life-long deformities only to be corrected by orthodontia. If these habits are corrected be-

fore the second year, Nature will overcome the majority of the cases of malocclusion, and the exceptional cases may require very simple treatment at that early period.

My experience has proved that regular, continued prophylactic care is the most valuable work done in the office. At each visit, every tooth is examined (1) for beginning caries; (2) for malocclusion, and (3) as to whether it is being used for mastication, and the parent's attention is directed to the defects.

Until we have had our patients return regularly every two months, we do not realize how little thought is given by the parent to the condition of the child's mouth and how surprised the mother is that so much time has elapsed since the last visit. This makes it necessary to have a regular system of calling the patients to remind them of the appointments, but it is a form of dentistry greatly appreciated by the parents, especially when the child leaves our care at the age of 15, with only a few small occlusal fillings and so educated to the appreciation of the value of his teeth that his home mouth hygiene will insure him much better health than the average child enjoys.

If you have been successful in educating the patient to be tooth wise, he has formed habits of home care that prompt him to brush his teeth before retiring as surely as he undresses before getting into bed. One of the rules we try to teach little children is always to brush the teeth before taking off the shoes.

When a child of 6 first comes under my care I have him learn by heart the following rules for brushing his teeth:

THE CORRECT WAY FOR THE CHILD TO BRUSH THE TEETH

Have two brushes, one for morning and one for night. Begin with the brush dry and use no paste nor powder, Always place the brush on the gums, brushing toward the cutting end of the teeth—that is *down* on the uppers and *up* on the lowers. This brushes the gums over the teeth and makes them hard and healthy. After this brushing, wet the same brush, using plenty of cold water and go over them again and again. When finished, hang the brush in the sun to dry. The next time use the other brush. The object in having two brushes is that one will always be dry to begin with. The teeth should be brushed *after* breakfast and *before retiring—every day.*

I expect all mothers to read and follow these rules in her home care of her children. If the patient shows signs of carelessness, and many of them do, I require him to return once a week for examination and recitation of the rules until better habits are formed. In conjunction with home oral hygiene, rules of diet are stressed.

Since beginning work for children, over thirty years ago, I have always thought that when we can control the diet, caries of the teeth will disappear. After all these years, I feel that we still have much to learn on the subject, but when young children have come under my care and they have been fed on a diet of largely fruits and vegetables, limiting the carbohydrates and using plenty of milk, the results have been most gratifying. I realize that following such a diet is easier in Southern California than in many places, but rapid transportation has improved conditions in all parts of our country.

PITS AND FISSURES

Under normal conditions, the first molars are well formed when they erupt during the sixth year and if the child is normally healthy, the grooves do not soften. Unfortunately, the curse of candy is one that we have not as yet and perhaps never will be able to overcome. Even a small amount eaten daily will settle in the grooves and pits in the par-

tially erupted teeth and soften the enamel until, in a few months, a fine explorer will detect a break. At the regular appointment for prophylactic treatment, this is discovered and marked on the chart. The child and the parent are warned of the danger to the teeth and a small percentage of these defects never develop any farther. At each visit, these marked places are examined, and when a cavity seems to have reached the dentin, it is time to fill it.

Mastication of hard and fibrous foods does much to protect the teeth after they are in occlusion, but during their eruption, they must be given extra care. When the lowers erupt first, it may be necessary to flow thin cement over the occlusal surface or, more frequently, I have the mother press a small piece of magnesium carbonate over each molar the last thing before retiring. A great effort is made to prevent the eating of candy and starches during these months, and frequently the child comes once a month for prophylactic care. The need of extra home care is stressed, brushing the teeth after each meal and rinsing the mouth with salt and water or some other mouth wash. The result of this care is sometimes a perfect set of teeth.

There has always been much discussion as to the material to be used in restoring the occlusal grooves in the permanent molars, and I have watched the work done by others and tried many methods that I have seen advocated, but I believe the only perfect filling is a carefully fitted gold inlay. As the technic has improved, there seems little reason to suspect that these fillings will not give good service for a long period of years. In my practice, this is universal and the time of one operator is entirely devoted to this work.

I believe that gold inlays are placed for younger children in our office than in most offices, and I realize that it is because a child who has been coming for prophylactic treatment for several years is under better control than one who goes for a restoration for the first time. Another reason is that the cavities are prepared before the caries has progressed far enough into the dentin to cause pain, and the third reason is that permanent teeth are much less sensitive while the roots are still growing.

It is not at all uncommon to put in occlusal inlays in the lower molars for a child of 7, but if there is a deep buccal groove with a break near the cervical end, we rarely place the inlay at that age. Instead, the occlusal and buccal cavities are prepared singly and filled with either white copper cement or copper amalgam for a few months.

In many mouths, where there are cavities in the permanent molars when the children are brought in because of pathologic conditions in the deciduous teeth, these cavities are partially prepared and filled with temporary cement over a pledget of cotton dipped in modified phenol and left a few days while the deciduous teeth are being repaired.

One of the greatest mistakes of the average dentist lies in filling one or two teeth and leaving cavities, however small, uncared for. One of my most rigid rules is: "The mouth must be treated as a whole." Every tooth must be put in a condition of usefulness or else extracted. This is carefully explained to the parents, and if they refuse to have the mouth put in a healthy condition, the work is not undertaken.

When the mouth is placed in a condition of repair, the deciduous teeth are treated and filled, the teeth are cleaned and free from stains, and home mouth

hygiene is thoroughly established before any permanent work is undertaken on the permanent teeth. When the mouth is prepared in this way, we begin the work of restoring the permanent molar.

We frequently prepare all the occlusal cavities as if for inlays and fill with white copper cement, charging a fee for the cement filling. If such a filling is replaced within six months, the amount paid for the cement filling is deducted from the price of the inlay. This practice works out much better than trying to place all the gold at once, as frequently the parent is able to pay for one filling a month. It does not tire the child to have an occasional filling, nor leave a sense of fear, as it would if several fillings were placed in succession.

On account of the difficulty of keeping children out of school, it is our practice to have the child excused from school for a half day. The cavity is prepared, and while the inlay is being cast, the child returns to the waiting room and amuses himself with a book or the toys that are kept for that purpose. He returns to the operating room for the setting of the inlay, then goes back to his book or toys. When the cement is sufficiently set, generally in an hour, he returns for the polishing, and is then sent home with the work finished.

It is very important in making gold inlays for children to restore the contour of the tooth, carving all the grooves to the anatomically correct ideal.

With the utmost care in the original preparation, it is often necessary to do minor carving and polishing during the first six months after the inlay is set. I have never been sufficiently optimistic to consider a gold inlay as finished when it is polished and the patient is dismissed. This is another reason that prophylaxis is valuable as it enables us to watch and correct these minor defects, and gives, I believe, a much longer life to the filling than if it had no later care.

A buccoclusal filling in a lower molar, as well as one in the distolingual groove in an upper, needs careful watching. If the least defect appears within a few years, an extension of the cavity is prepared and filled with a small inlay, for which a nominal fee is charged. These weak points are less easily protected in a mouth of a child given to candy eating.

A candy-eating child nearly always develops tiny cervical cavities in the mesial surface of one or more of his first molars. This is not surprising, as the distal surface of the second deciduous molar is broad and flat, with no real contact point, making an ideal hiding place for bacteria. These points are carefully watched at each appointment for prophylactic care, and if such a cavity is detected (often through the assistance of a roentgenogram), the distal surface of the second deciduous molar is widely cut away and a small cavity is prepared in the permanent molar. No attempt is made to extend for prevention and the cavity is filled with copper amalgam. The deciduous cavity is prepared and also filled with copper amalgam at a subsequent appointment after the filling in the permanent molar has set.

The patient is instructed to return as soon as the deciduous molar becomes loose if it is not evident at the appointment for prophylaxis, so that an inlay may be put into the mesial portion of the permanent molar before the bicuspid erupts enough to interfere with the cervical preparation for the inlay. We call these hurry-up cases and impress on the patient and his parents the value of conserving the tooth by placing a filling at this time so it will not be necessary to cut through the marginal ridge of the crown to place

a mesioclusal filling. Also, before the bicuspid erupts, the cervical margin can be more carefully prepared and the filling better polished. In many cases, only a few days after the deciduous molar is lost, the bicuspid has erupted so far that there is not access to prepare for gold.

We have found that inlays for Class 2 cavities are better prepared by the indirect method as the cervical margins are more successfully adapted.

HYPOPLASTIC TEETH

In this day of increased speeding up of life and after a generation of carbohydrate excesses, a surprisingly large number of mouths showing hypoplastic teeth are presented to the dentist.

The patient is very fortunate if he is in the hands of a competent dentist at the time of the eruption of the first permanent molars as there is greater hope of these malformed teeth being conserved for a long life of usefulness.

I believe that G. V. Black proved to the profession that this condition is not the result of syphilis, but we still encounter "doubting Thomases" who have closed their eyes to other causes and class any abnormality as syphilitic. I must not forget to mention that several children in my care who were congenitally syphilitic have beautifully shaped, strong permanent molars. I doubt that hypoplasia results from specific infection within at least one generation.

I have a great many data that connect hypoplastic teeth with endocrine insufficiency, but every time I think that I have proved the case, some new case seems to disprove it. Calcium deficiency, whether caused by nonfunctioning endocrines, by illness of the expectant mother or even by the inadequate diet of the mother, is apparently the cause of the trouble. I have studied the history of a large number of such cases and always find that the trouble originated before birth. Dr. Bunting once wrote me that in the examination of skulls of premature babies, he had not found any deposit of lime salts in the matrices of the first permanent molars, so it must be that the injury is due to a disease of the matrix itself.

Protecting these deformed, defective teeth from caries is one of the most difficult pieces of work confronting the dentist because of the age of the child and the hypersensitive condition of the molars. The following method has met with success in my office:

First, dry and paint each molar with nitrate of silver followed by eugenol. At the next appointment, remove caries and fill each pit with copper amalgam, making no effort to extend the cavities to include all the pits. If not already a prophylaxis patient, the child is to be placed on that list, and at every appointment, any margin of a filling or any new cavity that may have developed is to be filled with the copper malgam. These fillings are to be left until the child is 15, when a half crown gold onlay is used to restore the tooth.

Masticating a diet of hard foods helps in preventing new cavities from forming in hypoplastic teeth.

PRENATAL CARE

If we are to insure better teeth for the future generation more consideration must be given the expectant mother. This naturally belongs to children's dentistry as it is for the protection of the unborn child.

During this period, monthly prophylaxis is of the greatest value and often prevents the development of pyorrhea and the destruction of tooth tissues that is so common in the mouth receiving no care.

Dental work during pregnancy can be undertaken with perfect confidence if a few simple rules are followed: 1. Never give any but the most temporary relief until the confidence of the patient is gained. 2. Give short appointments so that the patient does not become exhausted. See that she goes home and rests after each appointment. 3. Do not undertake any permanent restoration until the gums and teeth are clean and healthy.

With these rules in mind, we have often put in a number of inlays during the period, knowing that the mother can give the time for the work better than during the months while nursing the child.

The pregnant woman often requires special instruction in the use of toothbrushes, astringent mouth washes and antiacids. I am very liberal in my advice as to the use of various forms of magnesia as well as salt and soda.

The diet of the pregnant woman is of great importance as it must include not only sufficient calcium to insure her own health and to safeguard her teeth, but also calcium to supply the developing fetus. The diet should include one quart of milk a day, to be used whole, skimmed or sour as fits the needs of the individual. Starches and sugars must be sharply limited and fried foods excluded from the diet.

The ideal diet during pregnancy and lactation should be plain, containing no highly spiced or fried or rich dishes. The bulk of the food should be vegetables, fruits, dark bread and milk. Leafy vegetables should be eaten twice a day, both cooked and in salads. Masticating rye crisp or melba toast made of whole wheat bread keeps the gums in a healthy condition and helps in digestion. Milk is to be substituted for tea and coffee, and if the latter are used, they are to be taken unsweetened.

I have found that women desirous of bearing healthy children with a prospect of good teeth are willing to sacrifice their own tastes or desires, and they often lament the fact that they are not advised during their pregnancy about the danger of too much sugar or the value of milk in the diet.

INFLUENCE OF THE ENDOCRINE GLANDS

Just how much influence the endocrine glands have in the development of a tooth is not thoroughly understood. We know that if the thyroid gland is not functioning, the calcium of the blood may be thrown off in the secretions. That the glands do not function may be due to disease or malnutrition, but it is a subject that needs much research.

I have studied many cases of hypoplasia, and a few where the permanent teeth were missing, and I have found in most of them a history of goiter or thyroid deficiency in the mother. In none of the cases studied was there any evidence of syphilis.

Quoting from Clifford Wright, an authority on the endocrines, who has examined several of the mothers and children under my care:

> It is of interest that both mother and child had poor teeth, and I have felt that poor lime metabolism can be found in families, for undoubtedly we see whole families who have poor teeth and other families who have good teeth, and those with good teeth may never have taken any precaution with their dental work. . . . For some time, I have been using parathyroid and lime together in the treatment of these cases. In girls, if they are of the ovarian type, I add to that one of the ovarian preparations, and in some instances, I have put in small does of anterior pituitary, for undoubtedly this gland controls in some way the bony metabolism.

In speaking of two women that I sent for examination, both mothers of chil-

dren whose anterior teeth as well as their molars were hypoplastic, Dr. Wright said: "Just what the basal metabolism rating of these two women was during their pregnancy, it would be hard to estimate. They may have had an active hypothyroidism at that time and not show it now."

The examinations of these mothers had upset my theory that hypoplasia of the teeth was due to nonfunctioning of certain glands, but if we had examined them during pregnancy, they might have proved it and so been added to my list of goitrous mothers with children having hypoplastic teeth, showing the effect of nonfunctioning of glands on tooth development.

IN CONCLUSION

Two questions asked by a school physician who examines large numbers of high school boys and girls each year are as follows:

1. Why do dentists advise gum chewing for children? If the child was given a sufficiently hard diet would he not need all the saliva furnished by the glands to prepare the food for the first step of digestion?

2. Why do dentists of apparently good judgment advise leaving broken teeth and roots in the mouths of school children? The Bible tells us, "If an eye offend thee, pluck it out." Why does not the same rule apply to a rotten tooth?

I leave these questions for you to answer as they have an important bearing on the period when the first permanent molar is such a factor in the health problem of youth.

Are we ever justified in leaving a pulpless first permanent molar which at any time may become a menace to health?

In all our work for children, is not our slogan "Health first"?

THE SCIENCE OF DENTISTRY*

By LEROY M. S. MINER†, D.D.S., M.D., Boston, Mass.

DURING the last few months, our attention has been turned back to the early days of the Massachusetts Bay Colony. The history and traditions of our grand old Bay State have been on every tongue.

These glances into the past have served to make us more conscious than ever before of the wonderful age in which we now live. That thing we call *science*, which has brought us the ability to fly, to talk through limitless space, and a thousand other miracles that we take more or less for granted, would probably in those earlier days have been called black magic or witchery. The achievements of science in every field of endeavor have brought about new modes of living, new comforts and pleasures, and new dangers as well.

The advances made in the science of dentistry have kept pace with those made in other branches and have been rapid

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and far-reaching, especially since the World War. Entirely new purposes and a new idea of the work of the dentist have appeared as a result of this new scientific development. In former days, dentistry was largely a mechanical vocation, tinctured with esthetics—or art, if you please. The pride of that day was mechanical skill. Today, dentistry is an important cog in the great health service machinery, which works to keep the health of the nation at its best, and has as its chief interest the prevention of disease. The modern dentist is therefore acutely conscious of his responsibility to build up and maintain the health of the people.

Before scientific dentistry became a fact, teeth were filled and treated without much thought that they were living structures, intimately connected with the rest of the body. To show how intimate this connection is: a solution of lead injected into the stomach of an animal can be detected in the teeth within thirty minutes.

Also, we now know that disturbances in the teeth may have profound and far-reaching effects on the whole body. Sir Wilfred Grenfell, in writing an appreciation of the work done by dentists in Labrador says, "The immense reduction in the arthritis cases and the other results of neglected focal infection, to say nothing of the improved metabolism from the care of children's mouths, have been one of the most satisfactory outcomes of our whole medical service."

In former years, the practicing dentist was content merely to remove decay from the teeth and fill the cavities so made with one of the several filling materials. Today, we study the patient with a view to finding the causes of decay and preventing its further development by the removal of those causes. To illustrate: A boy of fourteen years comes to the clinic at the dental school. He is considerably overweight for his age, fat and flabby. Examination of his mouth shows it is dirty; there are a dozen or more decayed places starting, where food débris has collected; the saliva is thick and stringy. We find in general that he is mentally and physically inactive. The dentist formerly would have been content to clean up the boy's mouth, remove the decay, fill the cavities, and regard the treatment as complete, with the chances that, in six months or a year, his mouth would again be in just as bad condition.

Today, a dentist, with the new idea of his part in maintaining health and preventing further disease, would recognize the high susceptibility to decay of the teeth in such a patient. The causes of this susceptibility are suggested, first, by the location of the decay in areas not swept clean by Nature's toothbrush, which are the muscles, saliva and coarse and rough foods, and, secondly, by the fact that this boy is at an age when growth is rapid and the demand for bone and tooth building material in his diet is great. Moreover, the thick, ropy saliva and excess weight suggest overeating of starchy foods, with too little of the bone and tooth building foods, a weakness thus developing in the structure of the tooth itself. This weakness may be a part of the adolescent period, or other physical reactions, but, more frequently than not, the fault will be found either in his selection of foods or the manner in which they are prepared, or perhaps may be laid to a faulty habit of sleep or a lack of exercise or fresh air, which may interfere with the proper benefit from his food.

Another type of case coming under my observation may be further illustrative. The patient was a girl of 8, underweight,

poorly nourished and pale. Examination of her mouth revealed the fact that all first permanent molars had come through the gum without any enamel on the chewing or grinding surfaces. Some of the front teeth had deep pits without enamel. Decay had started. What should be done? Advice had been given to extract the four molars. The child's history revealed the fact that at about 2 years of age, she had had several of the children's diseases and was for some time undernourished. It was suggested that by proper diet and the use of certain substances rich in vitamins and minerals, decay might be controlled. For six years, the mother followed carefully the advice given, and at the end of this time, when the patient was examined, no decay at all was found. The teeth had hardened, no fillings were necessary, and even the molars, bare of enamel, were as hard as enamel itself. These two cases illustrate what the new science of dentistry is, how it differs from the old mechanical dentistry and what it can do to preserve your health.

Cleanliness is, of course, always a help in maintaining a healthy mouth, but it does not of itself prevent tooth decay. Important scientific researches have shown that the causes of tooth decay go far deeper than mere lack of cleanliness in the mouth. The search for the causes of tooth decay—a disease so widespread that it has often been called the "people's disease"—has approached the point where we feel that a lack or deficiency of certain substances in the blood favors the breakdown of the teeth by decay. Intensive scientific work is therefore being done, with the hope and expectation that tooth decay may eventually be controlled, and no longer be a curse to the human race.

Similarly, that disease of middle life which we call pyorrhea, so long regarded as merely a disease of the mouth alone, and treated as such, is being also critically studied, and results again suggest that changes in the physical condition of a person are a matter of considerable importance in the first development of the disease.

As a matter of fact, the soft tissues of the mouth are extremely delicate, and frequently are a barometer of a person's physical condition. If one part of a body suffers, all parts suffer indirectly, and disease in any part of the body is likely to show its effects on the tissues of the mouth. The late Sir William Osler, one of the world's most famous physicians, has said, "There is not any one single thing more important to the public, in the whole range of hygiene, than the hygiene of the mouth." To that statement might be added that the mouth is the gateway to the body, and that gateway must be kept in good condition.

In the efforts being made to improve the dental health of the nation, the children have received chief attention. Through the munificent gifts of the Forsyths, Mr. George Eastman, and others, it has been possible to attack this problem in a concentrated fashion.

In studying not only the mouth of a child, but also the child as a whole, we find a number of interesting things taking place. Many mothers are interested in the question as to whether or not the first baby teeth are of importance, since, sooner or later, they are lost and are replaced by the permanent set. Much emphasis is now being placed on the care of these temporary teeth, and the importance of maintaining even the temporary teeth in healthy condition is now widely recognized by the dentist and should be by the general public.

When care is not taken of the baby teeth, a definite procession of serious results will occur. Starting with decay in a temporary tooth, the first thing that happens is that it becomes sore and painful to bite on, and the child therefore does not like to chew his food. Then two things happen: either he bolts his food without chewing it at all, or the food is washed down with water or milk, or other liquid. As a result of the lack of use of the teeth, we find that the bones, not only of the jaws, but also of the whole head, fail to develop completely. They are undersize, and so, when the second teeth erupt, there is not enough room for them, with the result that they overlap and are crooked and unsightly. Teeth in this position are much more likely to decay, and then the whole series of infections which come with these secondary teeth and other effects on the body have their beginning.

As a result of lack of complete development in the head, we get constricted breathing spaces. The child cannot breathe as fully as he should through his nose, so he breathes with his mouth part way open, and the mouth breathing habit is established. Incomplete respiration and lack of function of the chest results in incomplete chest development, and, by this time, we have a substandard child.

We see, in some children, the effects in the digestive system from lack of chewing and washing down the food with liquids. Food is not converted into energy and new tissues as it should be. Food is not utilized, that is, not completely changed into energy. This we call malassimilation, which is the forerunner of undernourishment, and when this develops, the child is a prey to all the infections and diseases of childhood.

It may be seen from this that to prevent decay of the teeth in the first place is of fundamental importance and an important factor in the maintenance of the health of the child. Public health and the health of the individual are the foundation upon which rest the happiness of the people and the welfare of the state. Health is our greatest asset, but we are apt to take it more or less for granted, and often fail to appreciate it until it is lost. We neglect to take proper care of ourselves; we break the laws of health and hygiene; we ignore known methods of disease prevention, and while for a time people escape paying the penalty, sooner or later Nature will resent abuse, will refuse to do its work properly, and then—gone is that abounding good health to which everyone is entitled.

Organized dentistry has for the most part been interested in efforts to improve the standards of the profession, but the vision of dentistry is now broader, and efforts are being made to give the public dependable information regarding the problems of mouth health. The public may expect a constantly increasing effort on the part of dentistry to eliminate dental disease.

THE DENTIST OF TODAY AND HIS RELATION TO THE PUBLIC*

By WILLIAM RICE†, D.M.D., Boston, Mass.

MARCONI has recently announced that radio waves not only encircle our globe in a few seconds and can be interpreted wherever there is a receiving instrument, but they also pass out from our planet to almost inconceivable distances. It is an appalling thought to one approaching the microphone that the voice is to be projected into space to an immeasurable distance and to be heard by a countless audience. The audience may indeed be "countless," for all listeners have the privilege of "shutting off" at any time.

The field of dentistry has broadened in its scope and the interest of the dentist is not limited to matters relating to the teeth alone. He is also concerned with the normal upbuilding of tissues of the body, in order that a state of health may be more surely maintained.

I think the public should know what it means in time and study to become a dentist today. In no other profession have the educational requirements increased with such rapidity. As recently as 1916, admission to the New England dental schools was based upon graduation from an accredited high school, while the course in dentistry was of three years' duration. Since that time the requirements have been increased on three different occasions, and at present the entering student must have completed two years of study in an accredited college of liberal arts, followed by the professional course, which is of four years' duration. In addition to this, many dental graduates are taking a year's internship in hospitals. The number of hospitals in which the dental intern is considered a necessity is rapidly increasing.

In view of this change, it is not strange that the wide-awake practitioner who has been in practice for a period of years is taking advantage of every opportunity to attend the extension courses in dentistry now being given by universities in several states, and graduate courses given by the dental schools; also study courses which are given in connection with state and other dental societies.

I would not belittle the splendid work which has been done by dentists of the past. There have been many men of marked individuality and strength of purpose. They possessed the spirit of the pioneer and his inflexible determination to accomplish what he had undertaken to do. They had little to guide them by way of precedent, and only the crudest of instruments and tools with which to do their work. It is due to the initiative of the early American dentist that American dentistry today holds first place in all countries of the world.

Many of the subjects recorded in the early literature of dentistry are the same as those in which the profession is now

*Broadcast over WEEI under the auspices of the New England Dental Society.

†Dean of Tufts College Dental School.

interested. The marked difference is in the treatment of the various questions. This is due to the great advance in the knowledge of the sciences upon which practice in all branches of the healing art is now based. To Louis Pasteur, the great chemist whose tireless investigations resulted in the discovery of the germ theory of disease, can be accredited more than to any other man the unprecedented progress in both prevention and treatment of disease which has taken place during the past fifty years. Dentistry with all other branches of medicine has shared in the benefit of his discoveries.

The dentist of today looks on himself not only as one responsible for the remedial and restorative treatment of the teeth of those seeking his services, but also as the guardian of the mouth, which is the entering portal for the germs of many diseases, and is frequently the seat of the scourge which is each year taking an increasing toll in lives, and under conditions pitiful to relate. I refer to cancer. The modern dentist has acquired thorough instruction and, by actual visual experience, sufficient knowledge of malignant disease to be enabled to recognize the danger signs. "Is he expected to treat cancer?" some one may ask. The answer is "No!" But of even greater importance than treatment is the early recognition of conditions which, if neglected, soon pass beyond the stage when treatment by any means would be without avail. The custom already established in dental practice of requiring regular and frequent examination of the teeth affords an unparalleled opportunity for the early detection of cancer of the mouth.

Students in dentistry now have the advantage of a thorough course in hygiene and public health, for the reason that such training broadens their field of usefulness as citizens, which is the chief purpose of a liberal education.

It is not strange that the public has not yet come to regard its relation to the dentist in its true light. The fault lies largely in the established customs of a large percentage of the dentists themselves—customs which savor of trade rather than the true professional relationship which should exist between doctor and patient. A large number of dentists still base their fees on the number and kind of fillings they have made, the number of teeth on an artificial denture, the number of teeth extracted and the materials used—items which should have little bearing on the value of the service to the patient. Examination and advice have been given without charge, which is entirely without justification, for nothing exceeds in value to the patient a thorough examination and an intelligent diagnosis of prevailing conditions. Serious infections menacing the health and even the life of the individual can be discovered only by an examination in which the resources of modern science laboratories must not be disregarded. When restorative work is to be done, it is greatly to the advantage of both dentist and patient to have study models made for careful consideration of the case, that an intelligent decision may be reached. These are matters of utmost importance, and it is almost inconceivable that the dentist should be willing so to belittle the value of his training as to give free examinations and diagnosis, or that a reasonable patient would expect it.

In daily practice, the dentist is time and time again asked to follow the dictates of the person seeking his service. If the training and experience of the dentist stand for anything (and I am sure they do), it should be his prerogative to make his diagnosis and indicate the line

of treatment which he considers necessary. Of course, the financial resources of the patient must be taken into consideration, and it is frequently impossible at the moment to proceed to carry out the line of treatment indicated. When all is favorable, the dentist—not the patient—should decide on the course to be pursued; and the advice of the dentist is as worthy of compensation as is the production of a filling, an inlay, or a denture. To the conscientious dentist, each case is a problem in itself, differing from all other cases.

Let us hope that the time is not far distant when a better understanding of the meaning of higher education, a better sense of the general fitness of things on the part of both dentist and patient, and a greater regard for truth, which comes from the serious pursuit of knowledge, will result in the strengthening of public confidence in the sincerity of purpose of the dentist and his desire to give the needed service, rather than to follow the line of action which will give him the greatest monetary return.

What are some of the essential qualities which the public has the right to expect the dentist to possess in a marked degree? They are many and varied. I will cite some of them:

1. He should be a man of steadfast character and a fortitude which holds him to high ideals—which will not permit him even in adversity to resort to lowered standards.

2. He should have a genius for human relations and an insight into the many conditions influencing human actions; the ability to estimate the reality of suffering and to differentiate between the genuine and the imaginary; sympathy for all suffering, whether mental or physical; patience which stabilizes temperament and thus prevents anger, which is the precursor of cruelty; the desire for service which impels obedience to the unenforceable laws of conduct; sincere consideration of the charges for service, taking into account all known circumstances.

3. He should possess mechanical sense, which is to a considerable degree intuitive and without which it is difficult to acquire that skill and deftness of touch which distinguishes the expert operator from the man of ordinary powers. It is this quality in combination with keen powers of observation which marks the skilled surgeon, either special or general.

4. He should have that type of education which liberates the mind and stimulates initiative in thought and action.

5. He should possess knowledge of the structure and function of the organs and tissues of the body, and a reverence for natural perfection which would make ruthless injury impossible.

6. He should have an appreciation of harmony of line and color that all artificial restorations may be true to art.

7. He should show strict regard for personal hygiene and cleanliness in the conduct of his practice.

The establishment of a stable practice is the result of long and persistent effort, and the possession of a reputation for sincerity of purpose and ability to achieve. I have known many instances of young men, particularly young married men, finding it hard—very hard—to stick to standards of behavior, which alone make it possible for them to be true to themselves, and by so doing retain their own self-respect. They see others in the same community practicing slovenly methods and charging fees which are seemingly low, but which are in reality high, for they rendered no real service. They see the indiscriminate public going from office to office in bargain-hunting fashion for dental service as though it were a mar-

ketable commodity to be bought at the market price. Some of these young men are discouraged almost to the breaking point. It is difficult to advise them, other than to say that which sometimes sounds like hollow mockery: "Stick to it! You'll win in the end!" The public should think about this.

CURRENT LITERATURE

EXTRACTS AND ABSTRACTS

The Teeth and the Stomach: The rôle of the dentist in the prevention or the amelioration of gastric conditions is discussed by Arthur F. Hurst and J. F. Venables (*Dent. Surgeon,* Feb. 28, 1931). The small intestine is protected, in its office of digestion and as the place where absorption of food takes place, against unsuitable and improperly prepared food and drink, by a double line of defense, the mouth and the stomach. In the first, food and drink are brought to body temperature, chemical elements are diluted and hard particles are crushed by the teeth and softened by the saliva. If, through haste, the food is not thus prepared for entrance into the stomach, the gastric mucous membrane is daily subjected to mechanical, chemical and thermal irritation. In addition, if pyorrhea, tonsillitis or sinusitis is present, infected material is constantly swallowed. Under these conditions, the gastric juice dilutes the chemical irritants, brings the food to body temperature, dissolves connective tissue, softens lumps of carbohydrate foods and, through its content of free hydrochloric acid, destroys streptococci from the mouth, throat and nose, as well as most of the organisms present in contaminated food and drink. In the normal stomach, therefore, germs are destroyed and the food is prepared for entrance into the small intestine. In the 10 per cent of persons in whom there is a deficiency of gastric acidity, the mucous membrane is irritated by food improperly prepared in the mouth, and an excess of mucus is secreted to protect the membrane. The mouths of the secreting tubules thus become blocked and little or none of the gastric juice reaches the lumen of the stomach, hypochlorhydria giving place to achlorhydria. In some otherwise healthy persons, achlorhydria may be due to a constitutional condition. The small intestine becomes irritated just as the stomach does in achlorhydria, so that duodenitis and enteritis may develop in addition to the gastritis. The increased alkalinity permits bacteria to flourish, and because the barrier normally offered by the gastric juice is down, colon bacilli, as well as organisms from the mouth, may attack the duodenum. Achlorhydria is present in many cases of appendicitis, gallbladder conditions, pernicious anemia, chronic cholecystitis, arthritis, asthma, goiter and many intestinal infections. And as pyorrhea plays an important part in chronic gastritis and achlorhydria, it has an indirect part in the causation of many of these conditions. Achlorhydria is always present in pernicious anemia and the associated disease of the nervous system, in which glossitis is frequently present. This soreness of the tongue is often ascribed by patients or even by dentists to the teeth. Recognition of its cause may lead to diagnosis of the systemic conditions responsible. There is much clinical evidence that dental infection is an important factor in the pathogenesis of gastric and duodenal ulcer in persons predisposed by a constitutional tendency to hyperchlorhydria. Treatment of pyorrhea and extraction of infected teeth play an important part in the successful treatment of ulcer and a more important rôle in the prevention of recurrence. Dental hygiene results in a decrease in the amount of infected material swallowed. Abdominal operations should be postponed, when possible, until dental sepsis is corrected.

Actinomycosis: A case of actinomycosis is reported by C. R. Forder (*Dent. J. Australia,* March 2, 1931). A woman, aged 30, of good physique, good general health and having excellent teeth, had a swelling of the maxilla above the apex of the left lateral incisor, with multiple fistulas discharging watery pus containing yellow granules. Examination of the

pus revealed *Streptothrix actinomyces.* The roentgen rays revealed a large area of bone destruction around the apex of the lateral incisor. The tooth looked dark and was painful, and there was a minute cavity in the lingual pit that extended to the pulp chamber, pus escaping through the aperture when the hole was enlarged and the root canal entered with a broach. The infected tissue was excised under conductive anesthesia and the apical third of the root removed. A guttapercha post was inserted in the root canal after surgical treatment had rendered the canal fit to be sealed. The cavity resulting from excision of tissue was irrigated with a 5 per cent solution of "Dakamin" and was then packed with neosilvol crystals. A dressing was placed over the wound, and the enlarged root canal was left open for drainage. Potassium iodid was administered three times daily in doses of 10 grains. On the sixth day, the root canal was cleaned of exudate, dried, sterilized, and the previously made guttapercha post was cemented in. The cavity of the tooth was filled with amalgam. Recovery was uneventful, and the patient was still in excellent condition after a year.

Involvement of Nervous System Following Extraction of Teeth: Bailey (*J. Nerv. & Ment. Dis.*, February, 1931) reports a case of a woman, aged 66, who was said by her daughter to have been perfectly well until February, 1929. At that time she had six teeth extracted. One week later, she had six more teeth extracted, and finally several days later, she had the remaining seven removed. On that day, when she came home, she had difficulty in speaking and moving the jaws and after about two more days she became speechless and developed difficulty in swallowing. This condition had persisted until April, 1930, without any marked change in symptoms. The patient was able to understand conversation perfectly and seemed of normal intelligence. She complained of severe pain on both sides of her face, especially on the right. She also complained of severe headache. A physician at that time made the following report: "The patient is unable to pucker her lips, and blows out a lighted match with great difficulty. She shows no weakness on closure of the eyelids on each side. The tongue can be protruded slightly but not beyond the lips. It shows marked atrophy along the edges, and fibrillary tremors are distinct. The soft palate is innervated imperfectly. The patient is entirely speechless and at most can make only a few unintelligible noises. Her understanding of speech is apparently normal. When a glass of water is given to her, she takes a little into her mouth and swallows it with great difficulty, while a great deal of the water escapes and leaks out over her lower lip. The contraction of the masseter muscles is feeble." The case seems to be the only one in which a bulbar palsy has followed the extraction of teeth.

MISCELLANY

BOOK REVIEW

Year Book of the American Pharmaceutical Association, 1927 and 1928. Volumes 16 and 17, containing the seventieth and seventy-first annual reports on the progress of pharmacy, and the roster, a list of former officers, the constitution, by-laws, articles of incorporation, code of ethics, history of funds, treasurer's report and roll of members. Corresponding to volumes 75 and 76 of the former proceedings of the American Pharmaceutical Association. Published by the American Pharmaceutical Association, Baltimore, Md.

This is a very pretentious volume of 1403 pages, containing the constitution and by-laws of the association, its proceedings and all the detailed information necessary to keep the literature of the subject up to date. As we glance over its pages, we are impressed with the mammoth work necessary to prepare this volume, and we commend the compilers for making such a work available to all of those who are interested in the subject.

NEWS

Iowa State Society Elects Officers: At the last annual meeting of the Iowa State Dental Society the following were elected as officers for the ensuing year: president, L. W. Snuggins, Des Moines; president elect, Herbert D. Coy, Hamburg; vice president, John J. Booth, Marion; secretary, John Scholten,

Cedar Rapids; treasurer, Frank Fourt, Fairfield.

Appropriation for Dental Work in Hawaii Increased: Miss Helen M. Baukin, supervising dental hygienist of the Department of Public Instruction in the Territory of Hawaii, reports that the budget of the dental division of the department was increased recently by $43,710 biennially. This increase means that there will be a budget for the next two years' work of $196,130, which will permit the employment of five additional hygienists and three additional school dentists by the supervisor, making a total of thirty full time dental hygienists and eight full time school dentists on the staff. The increase in the budget was particularly gratifying in view of the fact that many budgets were greatly cut or set aside entirely.

Cleveland Dental Society Elects Officers: The following were elected officers of the Cleveland (Ohio) Dental Society at the annual meeting held recently: vice president, H. Steurer; president elect, H. Van Valkenburg; secretary, H. J. Hoppe; treasurer, G. L. Drach. C. J. Stark was installed as president.

Simplified Practice Recommendation on Dental Plaster: Simplified practice recommendation R117-30, on packaging of dental plaster, investment and artificial stone, is now available in printed form, according to an announcement by the division of simplified practice of the National Bureau of Standards, Washington, D. C. Copies of this recommendation, which has been instrumental in reducing the number of package sizes of dental plaster from twenty-two to three, and dental investment from twenty-six to thirteen, can be obtained from the Superintendent of Documents, Government Printing Office, Washington, D. C., for 5 cents each.

Deaths

Frankel, James D., Chicago, Ill.; Northwestern University Dental School, 1907; died, June 6.

Vieweger, Paul T., Greenville, Texas; Texas Dental College, Houston, 1911; died, May 27, after an operation for appendicitis; aged 42.

ANNOUNCEMENTS*

CALENDAR OF MEETINGS

American Dental Association, Memphis, Tenn., Oct. 19-23, 1931.

American Dental Hygienists' Association, Memphis, Tenn., Oct. 19-23, 1931.

American Dental Assistants Association, Memphis, Tenn., Oct. 19-23, 1931.

American Dental Society of Europe, Paris, France, July 29-August 1, 1931.

American Society of Oral Surgeons and Exodontists, Memphis, Tenn., Oct. 16-17, 1931.

International Dental Congress, Paris, France, Aug. 3-8, 1931.

International Orthodontic Congress, London, July 20-24, 1931.

International Stomatologic Congress, Budapest, Sept. 2-7, 1931.

National Association of Dental Examiners, Memphis, Tenn., October 17.

Committee on Costs of Medical Care, Washington, D. C., September 17-24.

Greater New York, New York City, November 30-December 4.

Montreal Dental Club, October 15-17.

New England Dental Society, Boston, Mass., October 8-10.

Society for Advancement of General Anesthesia in Dentistry, New York City, third Monday in February, April, October and December.

Southern Society of Orthodontists, Cincinnati, Ohio, December 1-3.

Southwestern Dental Society, Albuquerque, N. M., October 13-15.

STATE SOCIETIES

District of Columbia, at George Washington University, Washington, D. C., second and fourth Tuesdays in each month from October to June, inclusive.

October

New Mexico, at Albuquerque (13-15).

*Announcements must be received by the fifth day of the month in order to be published in the forthcoming issue of The Journal.

December

Ohio, at Cincinnati (1-3).

MEETINGS OF STATE BOARDS OF DENTAL EXAMINERS

Montana, at Helena, July 13-18. T. P. Regan, Secretary, Helena.

North Dakota, at Fargo, July 14-17. Larry B. McLain, Jamestown.

OHIO STATE DENTAL SOCIETY

The sixty-sixth annual meeting of the Ohio State Dental Society will be held at the Netherland Plaza Hotel, Cincinnati, December 1-3.

EDWARD C. MILLS, *Secretary*
255 East Broad St.,
Columbus.

SOUTHERN SOCIETY OF ORTHODONTISTS

The Southern Society of Orthodontists will meet in Cincinnati, December 1-3.

SOCIETY FOR THE ADVANCEMENT OF GENERAL ANESTHESIA IN DENTISTRY

The next meeting of the Society for the Advancement of General Anesthesia in Dentistry will be held at Saltzman's, 60 E. 42nd St., New York City, October 26, at 7 p. m.

LEONARD S. MORVAY, *Secretary,*
76 Clinton Ave.,
Newark, N. J.

MONTANA STATE BOARD OF DENTAL EXAMINERS

(Change of Dates)

The Montana State Board of Dental Examiners will meet, July 13-18.

T. P. REGAN, *Secretary,*
Helena.

NEW ENGLAND DENTAL SOCIETY

The thirty-seventh annual meeting of the New England Dental Society will be held at the Harvard Dental School, Boston, Mass., October 8-10.

GEORGE F. BERSE,
1374 Massachusetts Ave.,
Cambridge, Mass.

GREATER NEW YORK DECEMBER MEETING

The seventh Greater New York December Meeting will be held at the Hotel Pennsylvania, New York City, November 30-December 4. This meeting is held under the auspices of the First and Second District Dental Societies of the State of New York. The Chicago Dental Society has accepted an invitation to furnish essayists and clinicians for the meeting. Plans are being formulated for a joint meeting of the medical societies of the five boroughs of Greater New York and the First and Second District Dental Societies.

JOHN T. HANKS, *Chairman*

AMERICAN DENTAL ASSOCIATION

Memphis, Tennessee. October 19-23, 1931.

HOTEL RESERVATION

In securing hotel reservations for the 1931 Session, consult the hotel rate-sheet and fill out the blank application below. Mail immediately to the hotel you wish to patronize. The hotel will then advise you of the reservation which has been made for you.

As your first choice may not be available, kindly indicate a second and a third choice. If none of these are available, the hotel manager will mail your application to the Chairman of Halls and Hotels Committee, who will make a reservation for you in as favorable a hotel as possible.

Please remember that a reservation constitutes a contract with the hotel to provide you with the accommodations you desire. If you find it impossible to carry out your part of the contract, namely, to occupy the room at the time agreed on, please write or wire the hotel, releasing it, in order that your room may be available for other members.

MAIL THIS APPLICATION DIRECT TO THE HOTEL

HOTEL RESERVATION

AMERICAN DENTAL ASSOCIATION, MEMPHIS, TENNESSEE, OCTOBER 19-23, 1931

.....Hotel. 1931

Memphis, Tenn.

Please reserve sleeping room accommodations as noted below:

.....Room(s) with bath for.....people. Rate desired $..... (per day)

.....Room(s) without bath for.....people. Rate desired $..... (per day)

Second choice hotel.....

Third choice hotel.....

ROOMS TO BE OCCUPIED BY: ADDRESS IN FULL

Applicant..... .....

....

....

Please confirm this reservation to applicant. I further agree to notify the hotel at once in the event I am unable to use this reservation.

IMPORTANT TO HOTEL MANAGER: In the event you cannot accept this reservation, please forward this application at once to Dr. E. F. Simpson, Chairman Halls and Hotels Committee, 735 Sterick Bldg., Memphis, Tenn., who will attend to the assignment of this reservation.

HOTEL	Single Room with Bath	Double Room with Bath	Single Room without Bath	Double Room without Bath
ADLER 73 Linden Ave.	$2.00 & up	$3.50 & up 5.00 & up (twin beds)	$1.50 & up	$2.50 & up
AMBASSADOR 347 S. Main	2.00	3.50	1.50	2.50
CATHOLIC CLUB 185 Adams			1.25	2.00
CHICKASAW 99 S. Main			1.00	1.50
CHISCA 270 S. Main	2.00 & up	4.50 & up 5.00 & up (twin beds)	2.00 & up	3.50 & up
CLARIDGE 109 N. Main	3.00 & up	4.50 & up		
DESOTO 154 E. Calhoun			1.25	2.00
ELK'S 69 Jefferson	3.00-3.50	4.00-5.00 5.00-6.00 (twin beds)	2.00-2.50 Con. bath	3.00-4.00 Con. bath
FOREST PARK 855 Union	2.00	2.50	1.00-1.50	1.50-2.00
GAYOSO 139 S. Main	3.00 & up	5.00 & up	2.00 & up	3.00 & up
HERMITAGE APTS. 200 Union	1.50	2.75	1.00	1.75
WM. LEN 110 Monroe Ave.	2.50 & up	4.50 5.00 (twin beds)		
LONGINOTTI S. Main St.			1.00	1.50
PARKVIEW 1914 Poplar	3.50	5.00-6.00		
PEABODY HOTEL 149 Union	3.50 & up	5.50 & up 7.00 & up (twin beds)		
PLAZA 214 Calhoun	2.00	3.00	1.50-2.00	2.50-3.00
POLK APTS. 630 Madison	2.00	2.50-3.00		
PONTOTOC 69 Pontotoc	1.50	2.00	1.00	1.50
TENNESSEE 80 S. Third St.	2.00 & up	3.00 & up		
WINONA 110 E. Calhoun	2.00	3.00	1.00-1.50	2.00-3.00
WALDORF S. Main St.	1.50	2.00	1.00	1.50

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